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HISTOPATHOLOGY OF THE BRAIN AND SPINAL CORD IN A CASE PRESENTING A POSTINFLUENZAL LETHARGIC ENCEPHALITIS SYNDROME

HENRIETTA A. CALHOUN, M.D.
Assistant Professor of Pathology and Bacteriology, State University of Iowa

IOWA CITY

The occurrence of small epidemic foci of epidemic encephalitis in Iowa during the spring of 1919 raises the question of a new disease entity that can reach its ultimate solution only by the publication of necropsy studies and careful pathologic examinations, bearing in mind always the toxic or infectious origin of the changes. Clinically it is not always possible to differentiate toxic ophthalmoplegia with lethargy from Heine-Medin's disease, or botulism with ophthalmic symptoms.

REPORT OF CASE

History.—Through the courtesy of Dr. C. P. Howard, professor of internal medicine, it is possible to present the case of F. T. (Clinical No. 6075), a man, aged 37, who in January, 1919, had an attack of influenza followed by pneumonia from which he apparently completely recovered. On March 25, he was admitted to the university hospital complaining of pain and weakness in the arms and legs. Following his admission he became progressively worse, developing a typical syndrome of lethargic encephalitis which included (1) sleeplessness followed by drowsiness with nocturnal delirium, that deepened into a stupor from which he could be roused to answer questions inteiligently; (2) cranial nerve involvement shown by early transient diplopia, ptosis, mask-like facies and difficulty in swallowing; (3) spinal cord symptoms shown by a late loss of knee jerks, incontinence of urine and feces and rigidity of the neck. Death occurred April 10, 1919.

Necropsy.—The brain weighs 1,530 gm. The dura is free, thin, with congestion of the blood vessels. The superior longitudinal sinus contains no thrombus. On cutting the dura, a large amount of cerebrospinal fluid escapes. The pia-arachnoid over the base and convexity is thin and transparent, with congestion of the pial vessels. It strips with ease. The convolutions are well formed but slightly flattened. In the left frontal region there is a pin-point cortical hemorrhage. The basal blood vessels show no sclerosis and are moderately filled with blood.

Longitudinal section through the middle of the thalamus shows the gray and white matter well differentiated. The lateral ventricles are normal in size.

The ependyma is smooth and there are no cysts in the choroid plexus. The blood vessels of the cerebrum, especially in the centrum ovale and the basal ganglions, are congested and the lymph spaces widened. The pons and medulla show congestion and hyperemia.

The cerebellum shows no gross pathology.

The blood vessels of the spinal cord, especially those over the dorsolumbar region of the cord, are markedly congested, showing distinctly through the dura mater. On section, the cord shows brownish areas bordering the lateral portion of the horn and in the posterior columns symmetrically placed; and reddish or pinkish areas extending in radially from the periphery of the cord. The differentiation between the gray and white matter is not distinct, and the color is a dirty gray (Fig. 1).

Specimens from the brain and spinal cord were hardened in alcohol or in 10 per cent. formaldehyd and stained with thionin, Mallory's phosphotungstic acid hematoxylin, Herxheimer's fat stain, hematoxylin and eosin and osmic acid.

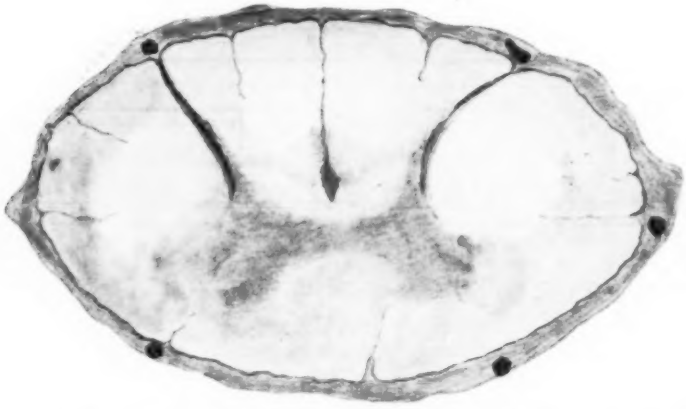

Fig. 1.—Dorsal region of the spinal cord showing a loss of differentiation between the gray and white substance, congestion of the pial blood vessels, and brownish areas of extravasated blood. Drawing from gross specimen, unstained; × 8.

Sections from different portions of the cerebral cortex stained with thionin show, especially in the frontal and anterior central convolutions, a disturbance of the cortical architectonics which is chiefly due to congestion of and infiltration about the blood vessels. There is an increase in the number of nuclei within the vessel walls, and a deposit of bluish staining granules. Yellowish pigment granules are also found either free in the vessel wall or deposited within epitheliod cells. With sudan III these granules are brilliant red. A few lymphocytes and plasma cells are present in the perivascular spaces. There is no new blood vessel formation. The marginal glia feltwork is denser beneath a slightly thickened pia-arachnoid, and there is an increase in the number of nuclei in the marginal zone. The nerve cells in the ganglion layers show a loss of the Nissl bodies. The cell outline is rounded, and the nucleus is often

ERRATA.—As will be noted, Figure 1 on page 2 was reversed in printing.

eccentric. The cytoplasm may show vacuolation or reticulation. Three to five satellite neuroglia cells are occasionally seen at the base of the large pyramidal cells.

Scattered irregularly through the cortex there are small, round, deeply staining masses of nuclear substance, which are about one third the size of a small neuroglia nucleus. In size and staining reaction they suggest neuronophages, but in their distribution they bear no relationship to nerve cells. A few rod cells are present (Fig. 2).

Fig. 2.—Section from the cortex showing nerve cell changes, satellite neuroglia, two types of neuroglia cells and the small, deeply staining cells which may be Maximow's polyblasts. Stain, thionin. Zeiss obj. D D; oc. 4.

In the white substance there is a deposit of granules in the vessel walls and some round nuclei in the perivascular spaces. One section shows a miliary hemorrhage at the cortical margin.

Sections through the basal ganglions show the blood vessels in the gray matter standing out sharply because of the presence of round, homogeneous, deeply staining bodies in the adventitia and in the perivascular spaces. There is also an increase in the cells in the vessel walls. Under the low power the

homogeneous bodies give the appearance of a perivascular infiltration of lymphocytes and plasma cells, but under the high power only a few cells are found (Fig. 3).

The nerve cells have lost their angular outline, are rounded and stain a diffuse blue with thionin. A few of the ganglion cells contain yellow pigment granules, and about some of them there are from 5 to 7 neuroglia nuclei. The small, deeply staining glial forms predominate over the large, pale nuclei. (Fig. 4).

The upper part of the pons shows the blood vessels sharply outlined by an infiltration of mononuclear cells and homogeneous bodies in the adventitia and perivascular lymph spaces in the gray nuclei of the pons. There are a few granule cells filled with yellowish pigment granules in the infiltration. The blood vessels are congested. In the lower portion of the pons there is a proliferation of the subependymal glia with irregularities in the outline of the ependymal glia lining. The blood vessels are prominent, congested, and the nuclei in the floor of the fourth ventricle show deeply staining pigmented cells. In one portion there is an extravasation of blood into the perivascular space (Fig. 5).

The blood vessels of the cerebellum do not show the infiltration, collection of homogeneous bodies, and congestion noted in the basal ganglions, pons and medulla. The cells of Purkinje are rounded in outline, with a homogeneous cytoplasm and a loss of almost all of the Nissl granules. At times the cytoplasm is reticulated. The nuclear membrane may be irregular in outline and the nucleolus stains very deeply. The cell processes may show more distinctly than normal (Fig. 6).

Sections from the cervical and upper dorsal regions of the spinal cord show a proliferation of the ependymal cells about the central canal, with a blocking and closure of the lumen. The glial septums are thickened as a result of both congestion of the vessels within the septums and a neuroglia proliferation. Along the lines of the septums are collections of homogeneous bodies and a few lymphocytes. In the white substance adjacent to the lateral horn there is an extravasation of red blood cells, some of which have disintegrated.

About the margin of the cord and scattered through the lateral columns there are spaces in the ground substance, where both the axon and myelin sheath have dropped out. These are the areolar placques, or sieve-like areas of an infiltrative myelitis. The spaces are sometimes filled with a pale staining, homogeneous substance. The ganglion cells in the anterior horn show chromatolysis, and the nucleus is displaced to one side, but there are no indications of neuronophagia, and no hemorrhages into the gray matter. The pia arachnoid is thickened and homogeneous in appearance, with few nuclei. The posterior roots show areolar plaques and many deeply staining homogeneous bodies. There are no secondary column degenerations.

A section of the lumbar cord shows the dura mater thickened and homogeneous, with a round cell infiltration about some of the blood vessels. The outer layer of the pia arachnoid is infiltrated with lymphocytes, while the inner layers are thickened, hyaline in appearance and closely adherent to the cord, especially in the region of the posterior roots. The posterior nerve roots show congestion of the blood vessels, and in the entrance zone there are sieve-like areas and many homogeneous bodies. There is proliferation of the ependymal cells with obliteration of the central canal (Fig. 7). The nuclei in the vessel walls, espe-

cially in the gray matter, are increased, and some of the endothelial cells show double nuclei. Small, deeply staining particles of nuclear matter resembling neuronophages are present in the anterior horn, but nowhere are they seen in definite relationship to the nerve cells. The ganglion cells show the same change seen at higher levels. At the top of one of the posterior horns there is a small collection of red blood cells. The glial septums are widened and in places hyaline in appearance, and there are a few red blood cells along some of the septums. There are areolar spaces and swollen myelin sheaths along the periph-

Fig. 3.—Perivascular infiltrations in the basal ganglions showing homogeneous bodies, endothelial cells and lymphocytes in the adventitial and perivascular spaces. Stain, thionin. Zeiss obj. D D; oc. 4.

ery of the cord and in the posterior columns. Some of the spaces are filled with a delicate, homogeneous substance staining a pale pink with eosin (Fig. 8).

Section through the cauda equina and the tip of the conus has a widened central canal, with proliferation of the ependymal lining and of the subependymal glia. The blood vessels about the central canal as well as those of the pia arachnoid and of the nerve trunks in the cauda are congested, but there is only slight infiltration (Fig. 9).

A section of a posterior root ganglion includes a large nerve trunk in which there is a calcareous deposit. There are also lime salt deposits in the connective tissue surrounding the nerve trunks. The ganglion cells show chromatolysis and loss of Nissl bodies.

Summary of Pathologic Changes.—This includes: Congestion and edema of both meninges and brain substance; acute encephalitis with perivascular infiltrations which are more marked in the basal ganglions and the nuclei of the pons and the medulla; nerve cell changes consisting of chromatolysis, cloudy swelling, and axonal reactions such as are found in the toxic states and with

Fig. 4.—Section through the basal ganglions showing prominence of the blood vessels due to deposits in and about the vessel walls, nerve cell changes and focal collections of round cells and homogeneous bodies in the interstitial tissue. Stain, thionin. Zeiss obj. A; oc. 4.

breaks in the continuity of the nerve fiber; alterations in the cells of Purkinje similar to those in fatigue and exhaustion; granular ependymitis of the fourth ventricle; acute diffuse infiltrative myelitis with extravasations of blood into the white substance; deposits of homogeneous bodies in the posterior roots, posterior root zones, and diffusely through the cord substance and perivascular collections of lymphocytes and plasma cells; closure of the central canal

throughout the entire cord, with widening at the tip of the conus medularis; nonsystemic areolar plaques; calcareous deposits in the posterior root ganglion; edema and thickening of the pia arachnoid with perivascular infiltrations of the pial blood vessels.

FINDINGS IN EPIDEMIC ENCEPHALITIS

The most constant findings reported in necropsy cases of epidemic encephalitis are congestion and edema of the brain and meninges. Microscopically, there is an infiltration of the adventitial and peri-

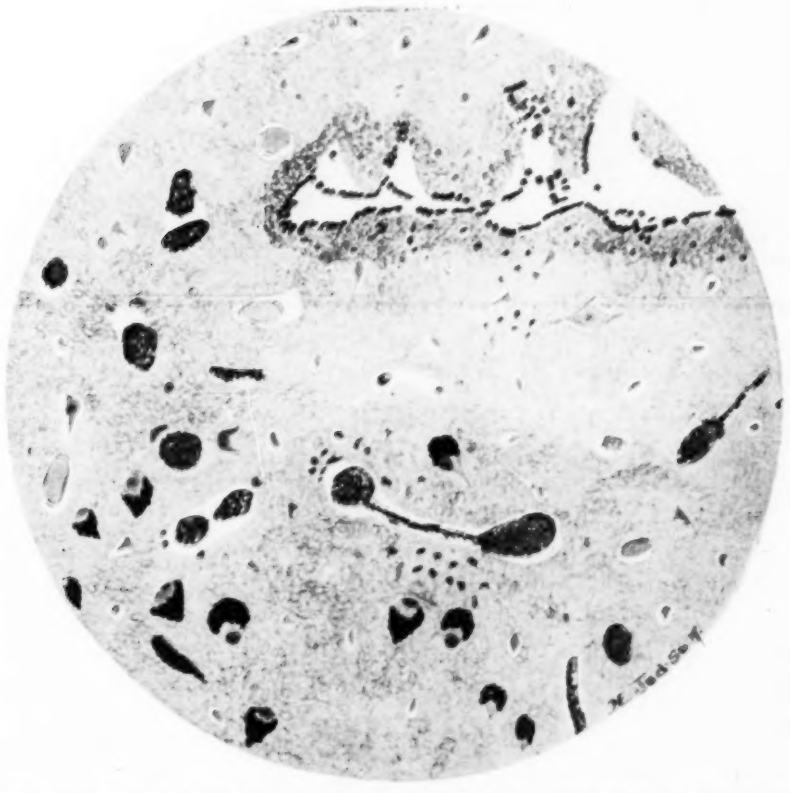

Fig. 5.—Section from the lower pons showing congestion of the blood vessels and granular ependymitis with proliferation of the subependymal glia. The nerve cells packed with pigment are part of the motor nucleus of the fifth cranial nerve. Drawn from a hematoxylin and eosin preparation. Zeiss obj. A; oc. 4.

vascular spaces with lymphocytes and plasma cells. These perivascular rings, occurring especially in the nuclei of the brain stem, have been considered almost pathognomonic. In the basal ganglion and the cranial nerve nuclei, the vessels stand out sharply differentiated from

the surrounding tissue by the ring of nuclei about a distended vessel lumen. The nuclei in the vessel walls show division forms and an increase in numbers, but there is no evidence of new vessel formation, and epithelioid cells either of vascular or glial origin are rarely seen. The neuroglia nuclei are moderately increased, but in no case is there a record of a double perivascular ring of which the inner circle represents the infiltrating cells and the outer the neuroglia nuclei gathering about the margin of the perivascular spaces. The character of the infiltrating cells varies. Bassoe and Hassen[1] report lymphocytes, plasma cells, polyblasts, fibroblasts and rod cells in the adventitial spaces, perivascular spaces and the adjacent parenchymatous tissue; Marinesco[2] finds plasma cells and lymphocytes with some vessels floating in a small pool of blood, and von Economo[3] shows the adventitial sheaths infiltrated with lymphocytes, leukocytes and plasma cells. The case of F. T. differs from those contained in other reports because of the slight cellular infiltration of the vessel walls, and the presence of masses of homogeneous bodies packed in the adventitia and in the perivascular spaces of the blood vessels of the cranial nuclei and the basal ganglions. These homogeneous bodies stain with thionin and cresyl violet. They are rounded and vary in size, and probably represent accumulations of degeneration products. Low and Mott[4] described them in a case of sleeping sickness due to trypanosomiasis.

In the spinal cord there are two forms of homogeneous bodies, one consisting of small, round, deeply staining masses found along the glial septums, in the posterior roots, the posterior root zones, and diffusely scattered in the white substance. They take a deep blue stain with hematoxylin, but do not stain with either osmic acid or sudan III. They have been reported as corpora amylacea or amyloid bodies (Marinesco) and were present in Breinl's[5] case of acute polio-

1. Bassoe, Peter, and Hassin, George B.: A Contribution to the Histopathology of Epidemic (Lethargic) Encephalitis, Arch. Neurol. & Psychiat. **1**:24 (July) 1919.

2. Marinesco, G.: Local Government Board Reports on Public Health and Medical Subjects. London. Report on an Inquiry into an Obscure Disease. Encephalitis Lethargica, N. S. 121, p. 47, 1918.

3. Von Economo, C.: Encephalitis Lethargica, Wien. klin. Wchnschr. **31**:850, 1918.

4. Low, George C., and Mott, F. W.: Examination of the Tissues of a Case of Sleeping Sickness in a European, Brit. M. J. **1**:1666, 1889.

5. Breinl, A.: Clinical, Pathological and Experimental Observations on the "Mysterious Disease." A Clinically Aberrant Form of Acute Poliomyelitis, Australian M. J. **1**:209 (March 16) 1918.

myelitis. The other homogeneous bodies are found in the widened spaces in the ground substance, chiefly of the lateral columns. They take a definite pink stain with eosin and a pale stain with thionin, the effect being that of a faintly staining exudate. Stains for bacteria demonstrate no organisms within this homogeneous substance.

The nerve cell changes are more or less uniform throughout the brain and cord. There is chromatolysis and an axonal reaction found

Fig. 6.—Section from cerebellum showing loss of Nissl bodies, chromatolysis and nuclear changes in the cells of Purkinje. Stain, thionin. Zeiss obj. D D; oc. 4.

in the ganglion cells. The early axonal reactions shown by certain cells in the cortex are of especial interest in connection with the myelitic changes. The cytoplasm may be reticulated, and there are slight variations in the nuclear staining reactions. Neuronophagia has been reported in other cases, but is not present in the case of F. T. The small, deeply staining cells scattered through the cortex bear no

relationship to the nerve cells. In both the cord and brain the ganglion cells show a satellite gliosis about the base of the cell, but in no instance are cells seen penetrating the nerve cell or within the nerve cell body. This, associated with the absence of hemorrhages into the nuclei and gray matter, makes an important pathologic differentiation from acute poliomyelitis of the Heine-Medin form. Marinesco emphasizes the constant finding in his cases of changes in the Purkinje cells in the cerebellum, which have already been described in this case. The picture is that seen in fatigue and shock.

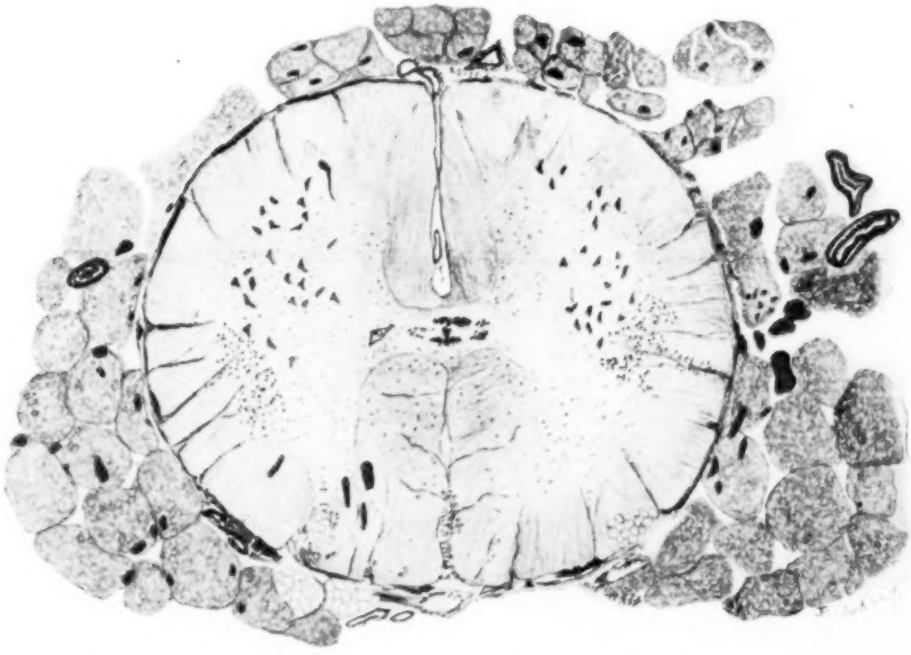

Fig. 7.—Section of lumbar cord showing closure of the central canal; areolar plaques along the anterior margin and in the posterior nerve roots; congestion of the blood vessels with slight hemorrhage into the white substance bordering the lateral horn; infiltrations in the lateral and posterior columns and subpial hemorrhages. Drawn from a hematoxylin and eosin preparation; × 8.

Spinal cord involvement was indicated by a loss of knee jerks and incontinence of urine and feces, other symptoms being masked by the coma. The diffuse nature of the process is shown by the dirty gray color of the cord with indefinite rusty discolorations in the white substance, which were present throughout the entire cord. Microscopi-

cally, the thickening of the neuroglia septums along which there are infiltrations of lymphocytes, plasma cells, and red blood corpuscles, the presence of an exudate in the spaces of the ground substance, congestion and perivascular infiltration, and the presence of areolar plaques and swollen myelin sheaths make a picture typical of that of an acute diffuse infiltrative myelitis. This picture is not new, Henneberg[6] giving a summary of the literature previous to 1911 on similar pathologic changes occurring after influenza. They are probably toxic in origin, the infiltration bearing a definite relation to the vessels arising from the vasocorona, the posterior and lateral vessels showing the greatest change. The thickening and slight infiltration of the pia-arachnoid of the cord presents a picture quite at variance with the severe, often purulent meningitis reported in cases of direct invasion of the meninges with B. influenzae.

No secondary degenerations are demonstrable by either Marchi or Weigert's myelin sheath stain. This is to be expected from the brief course and duration of the spinal cord involvement. The initial symptoms of pain may have been of root origin, judging by the collections of homogeneous bodies and the presence of areolar plaques in the posterior roots and posterior root zones.

The ventricular system, including the central canal, shows alterations. The ependymal lining of the fourth ventricle is broken and irregular, with proliferation of the subependymal glia. Granular ependymitis of this type, however, is not uncommon in cases coming to necropsy after 30 years of age. It is chronic rather than acute. The definite proliferation of ependymal cells about a closed central canal, the presence of a slight glial reaction, and the congestion of the neighboring blood vessels, together with the widening of the lower end of the central canal (Fig. 9), must be considered with reference to the theory that the virus of poliomyelitis is carried throughout the cord by way of the central canal, but the changes are so slight in the central gray matter, as compared to those in white cord mantle, that it seems improbable in this case for the spread to have occurred through this portion of the cord. No organisms can be demonstrated in the lumen. Bassoe and Hassin suggest that the amorphous mass in the closed central canal in their case may be made up of bacilli. Additional

6. Henneberg, R.: Die Myelitis: Handbuch der Neurology (Lewandowsky) **2**: Spezielle Neurologie I, 727, 1911.

necropsy studies are reported by Pothier,[7] Wilson,[8] Bassoe,[9] Netter,[10] Buzzard,[11] McCaw and Stebbing,[12] Gordon,[13] Mott,[14] Marinesco[15] and Marie.[16]

ETIOLOGY OF LETHARGIC ENCEPHALITIS

Of the etiology there is little that can be said. Undoubtedly acute infections, especially pandemics such as the influenza in 1918-1919, are frequently followed by the lethargic encephalitis syndrome. Zuelzer[17] talks of the sleepy sickness in 1712, Longuet[18] reviews the Italian epidemic of nona, and Leichtenstern[19] gives a complete summary of the literature on the comatose form of influenza. The case of F. T. and others recently reported differ from the true influenzal encephalitis in the absence of thrombi, softenings, abscesses and organisms. In true influenzal encephalitis Pfeiffer's bacillus has been isolated from the areas of softening and hemorrhage, the cerebrospinal fluid and the meninges by Nauwerck,[20] Trouillet and Esprit,[21] and Pfuhl.[22] In the

7. Pothier, O. L.: Lethargic Encephalitis, J. A. M. A. **72**:715 (March 8) 1919.

8. Wilson, S. A. Kinner: Lethargic Encephalitis, Lancet, **2**:7 (July 6) 1918.

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present epidemic of encephalitis no organisms have been found, if we except von Wiesner's[23] as yet unsubstantiated work, and no one has been able to transmit the disease to monkeys. It may be due to a toxin produced elsewhere in the body by the organism causing influenza, which may not be Pfeiffer's bacillus, or it may be due to an invasion by a new organism scattered to new localities by the world-wide movement of peoples. In the latter event the influenza pandemic by lower-

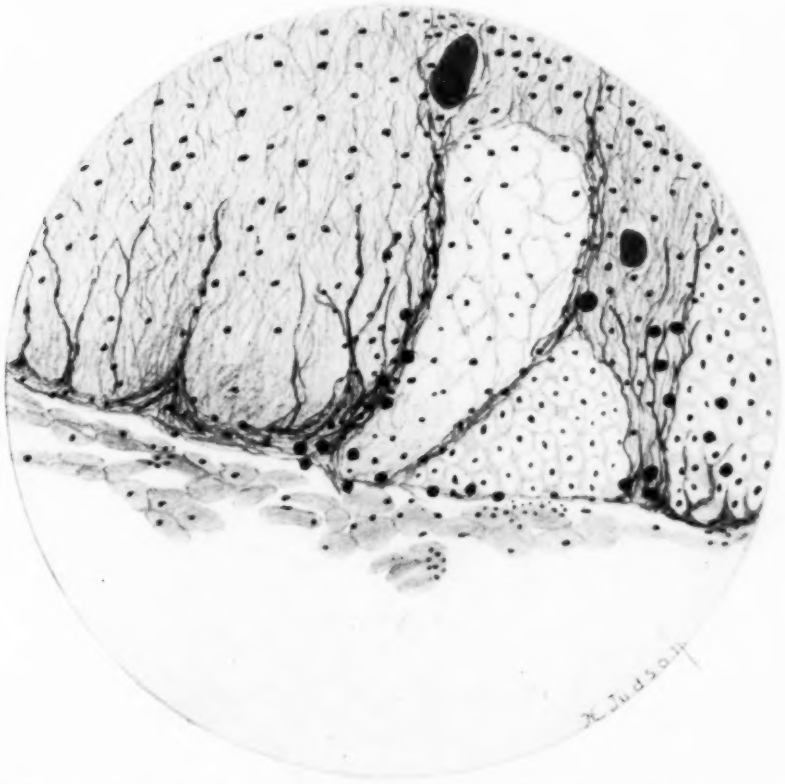

Fig. 8.—Section of cord showing a posterior root with areolar plaques, large purplish pink homogeneous bodies, small, deeply staining homogeneous bodies, thickenings of the pia-arachnoid and the marginal glia feltwork and congestion of the vessels in the cord. Drawn from a hematoxylin and eosin preparation. Zeiss obj. A; oc. 4.

ing resistance acts merely as a contributory factor. The same pathologic changes are found in trypanosomiasis and in the spirochete

23. Von Wiesner, R. R.: Die Aetiologie der Encephalitis lethargica, Wien. klin. Wchnschr. **30**:933, 1917.

infection of general paralysis; the changes differ only in degree and in location.

The combination of lethargy with cranial nerve involvement depends not on a specific virus, but on the location within the brain of the lesions produced by varying causes. Mauthner[24] localizes the sleep center in the floor of the fourth ventricle as a result of the findings in cases of nona, but MacNalty[25] explains lethargy and sleep as

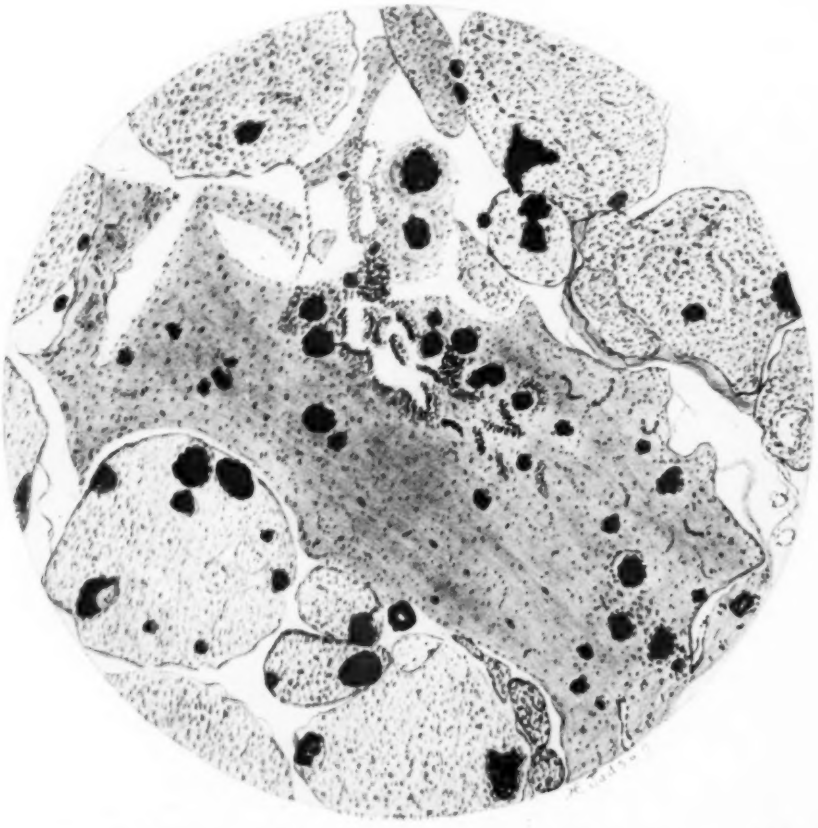

Fig. 9.—Section through the tip of the conus medullaris showing widening of the central canal, round cell infiltration, with congestion of the blood vessels of both the conus and the nerves of the cauda equina. Drawn from a hematoxylin and eosin preparation. Zeiss obj. A; oc. 2.

24. Mauthner, L.: Zur Pathologie und Physiologie des Schlafes nebst Bermerkungen über die "Nona," Wien. med. Wchnschr. **40**:961, 1890.

25. MacNalty: Local Government Board Reports on Public Health and Medical Subjects. London. Report on an Inquiry Into an Obscure Disease, Encephalitis Lethargica, N. S. 121, 1918.

due, not to a special sleep control center, but to an interruption of the sensory stimuli on the way up to the cortex by lesions in the thalamic region. This shutting off of the outside world reproduces to a pathologic degree the mechanism of normal sleep. Undoubtedly the same syndrome is clinically given by some cases of acute polioencephalitis and botulism. The epidemic in Queensland and New South Wales summarized by Breinl[5] and the cases reported by Mills and Wilson[26] are undoubtedly poliomyelitis with the lesions in the basal ganglions and the cranial nerve nuclei. They are differentiated from this case and the cases variously reported as lethargic encephalitis, epidemic encephalitis, nona, X disease, and toxic or epidemic ophthalmoplegia with lethargy, by the presence of hemorrhages into the gray matter and marked neuronophagia, and by the fact that the virus is transmissible to monkeys in the same manner as that of poliomyelitis. There seems to be at the present time in various parts of the world epidemic foci of Heine-Medin's disease and of epidemic encephalitis which clinically present the common syndrome of ophthalmoplegia and cranial nerve involvements associated with a progressive lethargy; pathologically, they are two distinct processes.

The lethargic encephalitis syndrome should be considered in the same light as Landry's paralysis or the Brown-Séquard syndrome — that is, as a localizing syndrome resulting from varying causes. The pathologic findings of epidemic encephalitis indicate that we are dealing with a new disease for this country, but the relationship to the pathology in trypanosome infections and spirochetal infections must be kept in mind in the search for an etiologic agent.

CONCLUSIONS

1. The term lethargic encephalitis is the name of a clinical syndrome caused by lesions of varying types which are localized in the basal ganglions and the nuclei of the pons and medulla. It is not a disease entity.

2. Included in this syndrome there are cases of epidemic encephalitis, a disease new to this country, whose etiology is unknown, but whose pathology bears a close resemblance to that of African sleeping sickness.

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26. Mills, C. K., and Wilson, George: Cerebello-Bulbar Polioencephalitis Originating During or After Epidemics of Influenza and of Poliomyelitis, Including the Record of a Case of Epidemic Encephalitis of the Lethargic Type, Arch. Neurol. & Psychiat. **1**:567 (May) 1919.

3. The case reported belongs to the new epidemic encephalitis group. The pathology is an acute infiltrative encephalomyelitis, the most marked changes occurring about the blood vessels of the thalamus, the cranial nerve nuclei, the floor of the fourth ventricle and in the white substance of the spinal cord.

A REVIEW OF THE EFFECTS OF GUNSHOT WOUNDS OF THE HEAD

BASED ON THE OBSERVATION OF TWO HUNDRED CASES AT U. S. GENERAL HOSPITAL NO. 11, CAPE MAY, N. J.*

CHARLES H. FRAZIER, M.D., AND SAMUEL D. INGHAM, M.D.
PHILADELPHIA

NEUROLOGIC ASPECTS

This report presents some general observations made in a series of 200 cases of wounds of the head, in practically all of which symptoms of injury to the brain was present. While the main purpose is to present a general survey of the cases as a group, certain aspects of the subject deserve more than casual mention.

Material.—These two hundred patients represent practically all of the cases of this type under observation at U. S. A. General Hospital No. 11, at Cape May, N. J., from October, 1918, to June, 1919. Of these patients, 88 per cent. suffered from wounds associated with demonstrable defects or fractures of the cranial bones, and 12 per cent. presented brain symptoms without demonstrable cranial injuries.

Patients with cranial defects	163
Patients with cranial fractures	13
No cranial injury evident	24

It will be seen that most of these patients presented cranial defects. These, with few exceptions, were the results of gunshot wounds of the head which were treated almost universally by early operation.

The associated brain injuries varied greatly, some being severe. In sixty-eight patients the cerebral symptoms were slight or could not be demonstrated when the patients were admitted to the hospital.

In 12 per cent. of the cases there was no definite history or evidence of cranial injury, but either general or focal symptoms of cerebral origin following traumatisms of the head seem to justify their inclusions in this series.

Symptomatology.—For the purpose of more systematic presentation, the manifestations of disturbed action of the nervous system have been classified into general and focal symptoms, in much the same sense that the symptoms caused by brain tumors are so classified; focal symp-

* Read at the annual meeting of the American Neurological Association held in Atlantic City, N. J., June 17-18, 1919.

toms representing lesions of definite areas of the brain, and general symptoms those resulting from the effects of diffuse forces, such as concussion or pressure.

EARLY GENERAL SYMPTOMS

Among the early general symptoms of importance, according to the clinical records and the histories as given by the patients themselves, were disturbances of consciousness, amnesia, delirium and confusion, choked disk, slow pulse, headache and vertigo. While obviously incomplete, these histories, considered collectively, have a certain value.

Disturbances of Consciousness.—Data concerning the state of consciousness immediately following the injury was available in 132 cases. In twenty-two there was no loss, while in the remaining 110 immediate unconsciousness resulted from the injury which lasted from a few minutes to several weeks.

TABLE 1.—PERIOD OF UNCONSCIOUSNESS

	No. Cases
No loss	22
Less than one hour	51
One to twenty-four hours	21
One to six days	22
Over six days	16

Since there was such a wide variation in the manifestation of this symptom it is of interest to consider the factors active in its production. These include: the degree of concussion, or sudden force transmitted to the brain by the blow; the amount of brain tissue traumatized, and the secondary effects of the injury, including hemorrhage, edema and infection.

For the purpose of analysis these patients may be roughly divided into three groups: (1) those in whom there was no loss of consciousness; (2) those unconscious from a few minutes to twenty-four hours, and (3) those in whom this symptom was prolonged.

Group 1 included twenty-two patients with cranial defects who were not rendered unconscious by their wounds. Many of these had severe injuries, and fifteen exhibited permanent focal brain symptoms. It is well known that a blow on any part of the head may produce unconsciousness by concussion, but it is apparent that this factor was insufficient to cause this symptom in the patients of this group. In explanation it may be suggested that the force of the injury was apparently exerted over a small area, and even when the cranial bones were fractured, and the brain itself traumatized locally, the diffuse

concussion must have been relatively slight. An illustration of this principle is furnished by the manner in which an egg may be broken, a quick, sharp blow producing a local fracture, while a slower but heavier blow results in extensive cracks in the shell. In the latter case the diffusion of the force is evidently greater than in the former.

Group 2 included seventy-two patients who were unconscious from a few minutes to twenty-four hours. It may fairly be assumed that cerebral concussion was the immediate cause of unconsciousness in this group, and that other factors were relatively unimportant in their

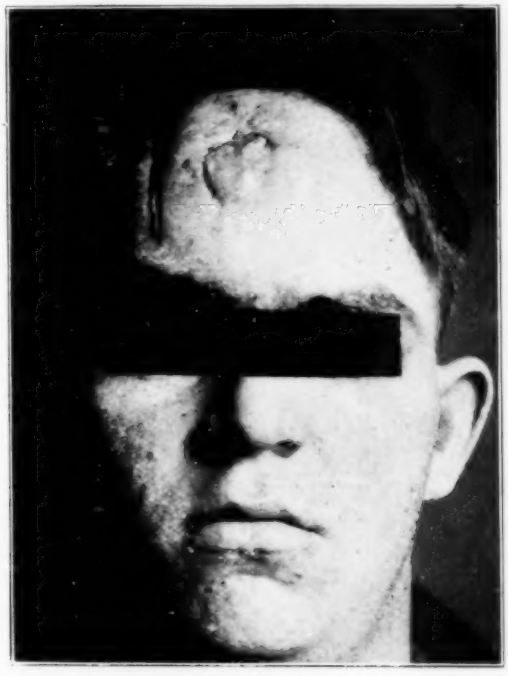

Fig. 1.—Patient with hernia at the site of the frontal defect.

effect on consciousness. Early surgical operations in many cases effected decompression, removed blood clots, pulped tissue, foreign bodies and bone fragments and controlled infection. It is probable that the character of the wounds themselves, in some instances, had the effect of automatic decompression, thus preventing prolonged unconsciousness.

Group 3 included thirty-eight patients who were unconscious for more than twenty-four hours. In many of these patients, in addition to cerebral concussion, it was evident that the secondary effects of trauma (hemorrhage, edema, infection) were important factors. Several of this group, with residual focal symptoms indicating severe

brain injury, had had early operations in which the dura was not opened, hence decompression not effected. Some had deeply penetrating foreign bodies, and others severe wound infections and hernia cerebri. In this class were also included ten patients with cranial fractures not decompressed. Even from the fragmentary records available, the large proportion of injuries not relieved by decompression was striking in this group, injuries which must have produced severe secondary effects and high intracranial pressure. In none of the cases was there evidence that prolonged unconsciousness resulted from concussion alone. While it is at times difficult to differentiate cases of uncomplicated concussion from those in which intracranial hemorrhage and edema are also present, it is apparent that prolonged unconsciousness resulting from simple concussion is rare. On the other hand, conditions producing increased intracranial pressure, such as hemorrhage and edema not relieved by decompression, must be considered as important factors in prolonging the unconsciousness primarily induced by concussion in head injuries.

Incomplete loss of consciousness, dazed and stuporous states, delirium and mental confusion were common in the early histories, one or more of these conditions frequently following the period of unconsciousness, or replacing it as the immediate effect of the trauma. These symptoms were regarded as results of the same factors that caused unconsciousness, concussion standing in relation to the earlier, and the secondary effects of trauma to many of the more prolonged manifestations. In this connection it should be stated that definite symptoms apparently resulting from simple concussion occasionally persisted for several months.

Amnesia.—Amnesia was present in practically all of the patients exhibiting the symptoms mentioned, and the memory blank frequently antedated the injury. In two instances patients who were injured in France had no memory of having been out of the United States. Those who were dazed or delirious for a long time often retained a fragmentary or dream-like memory of isolated occurrences, or of their subjective mental processes at times fantastic and curiously related to actualities.

Headache, Vertigo, Choked Disk and Slow Pulse.—These symptoms were recorded with varying frequency, and were all more or less closely related to the secondary effects of injuries.

LATE GENERAL SYMPTOMS

When coming under observation in Hospital No. 11, two months or more after receiving head wounds, many patients still manifested cerebral symptoms of a general character. These included loss of

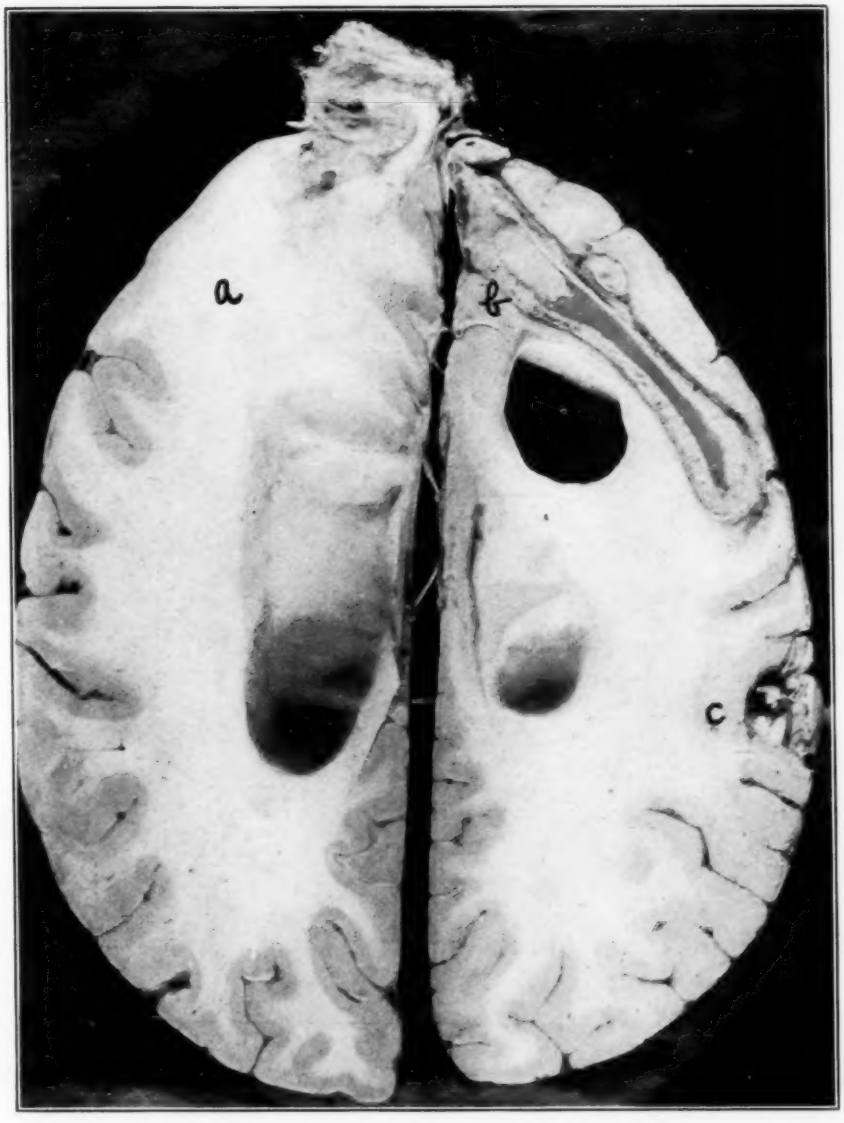

Fig. 2.—Brain showing (*a*) enlargement of left hemisphere and hernia cerebri at site of cerebral defect; (*b*) horn of dilated ventricle in relation with abscess cavity filled with inspissated pus; (*c*) bullet just beneath the cortex. Note relation of abscess cavity to trajectory between site of hernia and location of bullet.

memory, slow cerebration, indifference, mild depression, inability to concentrate, fatigability, nervous irritability, vasomotor and cardiac instability, general convulsions, fine tremors, irritable reflexes, headache, vertigo and restricted visual fields, but their manifestations varied in different patients as regards grouping, intensity and persistence. Some of them were present in most of the cases of severe head wounds, many of them were present in some of the cases, and exceptionally, a combination of these late general symptoms constituted the principal disability of the patient.

Almost without exception these symptoms diminished gradually, and ultimate recovery, apparently complete, occurred in from three to nine months after injury, where gross damage to the brain was absent. The tendency to recover from the symptoms, both general and focal, resulting from brain injuries of all degrees of severity, deserves special emphasis. Since it is fairly well established that regeneration does not occur in the central nervous system, it is evident that any nervous tissue may be affected to the extent of suspended function without suffering permanent damage, and recovery from the symptoms of brain lesions signifies returning function in injured but not devitalized neurons.

FACTORS CAUSING RESIDUAL GENERAL CEREBRAL SYMPTOMS

So far as could be determined, the following factors were operative in causing the late general symptoms in the series of cases under discussion:

1. Loss of cerebral tissue.
2. Injury to the brain without destruction of tissue.
3. Cranial defects.
4. Cicatrices.
5. Psychoneurosis.

Loss of Cerebral Tissue.—Symptoms resulting from the loss of cerebral tissue should properly be classified as focal, but these symptoms at times included intellectual impairment, or dementia, of which we have no definite knowledge in cerebral localization. Reference will be made, under the discussion of focal symptoms, to several instances in which the dementia apparently bore some relation to the location of the cerebral lesion.

Injuries to the Brain Without Destruction of Tissue.—These injuries include the effects of concussion and pressure, and also those of disturbed cerebral circulation and nutrition. Although, from the standpoint of pathology, changes of this nature are but imperfectly understood, it should be emphasized that they are common and impor-

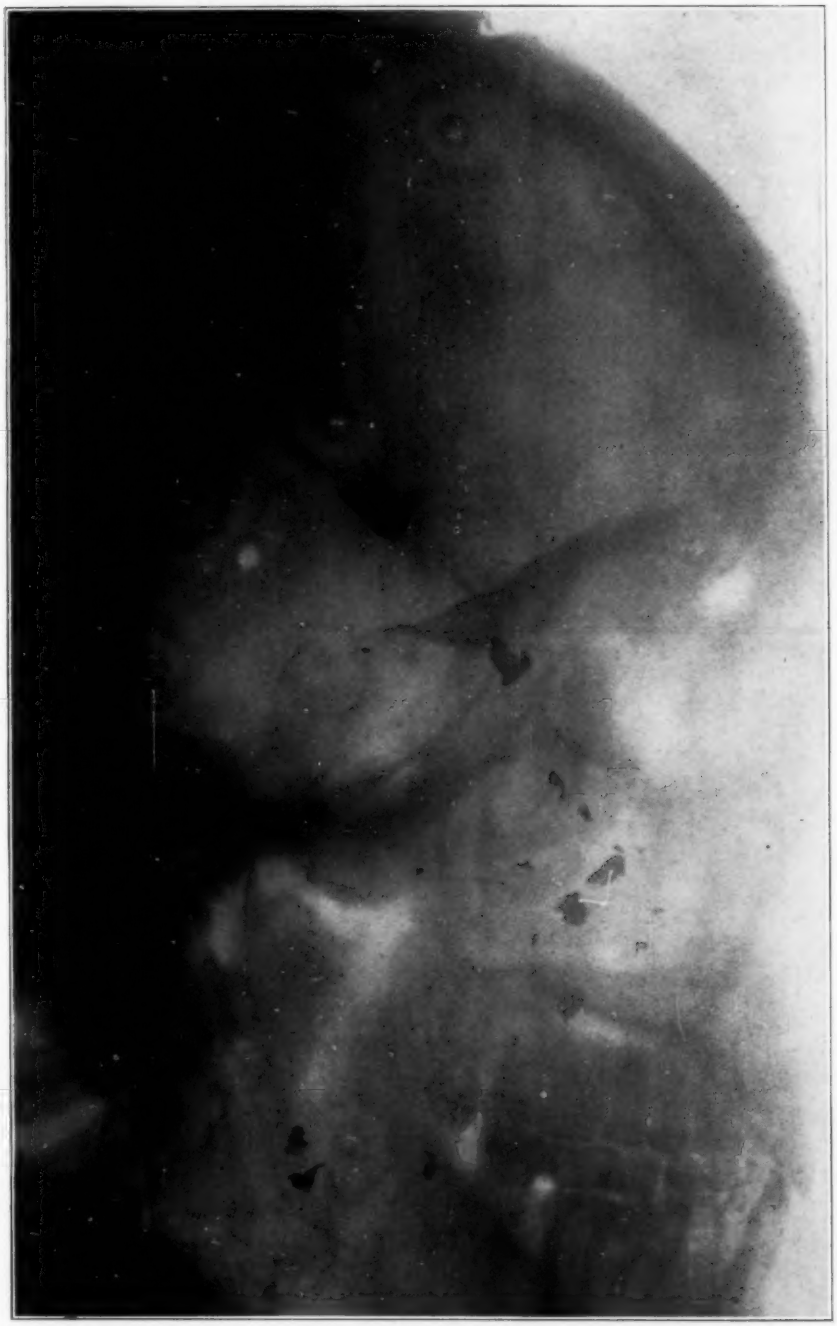

Fig. 3.—A "shower" of metallic fragments partly intracerebral and partly extracerebral.

tant. Most of the late general symptoms of head wounds are best explained on the basis of such disturbances. These symptoms include memory loss, slow cerebration, indifference, incapacity for sustained effort, vasomotor and cardiac instability, etc.

Cranial Defects.—These defects, particularly those large enough to permit fluctuation and pulsation, are commonly accompanied by vertigo, throbbing in the head and a feeling of insecurity, all of which are accentuated by active exercises and bending movements of the body. Headache, on the contrary, was noticeably unusual in the patients with cranial defects.

Cicatrices.—These sometimes act as irritating foci, causing nervous and reflex irritability, at times apparently precipitating general or focal convulsions. Headaches were often traced to pericranial and dural adhesions.

Psychoneurosis. — As an element in the symptomatology of this series this condition was comparatively unimportant. With three or four exceptions, anxiety and neurasthenic symptoms were present only to a degree commensurate with the nature of the injury. Conversion hysteria was not encountered in any of the cases.

Summarizing briefly the general cerebral symptoms resulting from wounds in relation to the etiologic factors, they may be divided into four groups: those due to (1) the immediate effects of the trauma; (2) the secondary effects of the trauma; (3) nondestructive injuries to cerebral tissue, and (4) destructive injuries to cerebral tissue. The first two of these groups of symptoms appear early, the latter two coming into prominence as the earlier symptoms subside.

The immediate manifestations consist mainly in disturbances of consciousness and in dazed, delirious and stuporous states, the principle causative factor being concussion.

The secondary effects of trauma (hemorrhage, edema, infection, etc.) add the symptoms of pressure to those of concussion.

Injuries to the brain tissue, not destructive in character, complicate all sorts of lesions, and cause symptoms which last for weeks or months, but which tend toward complete recovery. The syndrome of cerebral concussion (early disturbances of consciousness and prolonged mental symptoms including loss of memory, indifference, incapacity for sustained effort, mental slowness, etc.) probably has its pathologic basis in changes of this character.

VARIOUS SYMPTOMS

Transient Focal Symptoms.—Although the records were incomplete, they indicated that a considerable proportion of the patients suffered from focal symptoms of a transitory character, which disap-

peared completely or almost completely within one or two months following the injuries. Symptoms of this nature are to be explained by local injuries to the brain of a degree insufficient to cause tissue destruction.

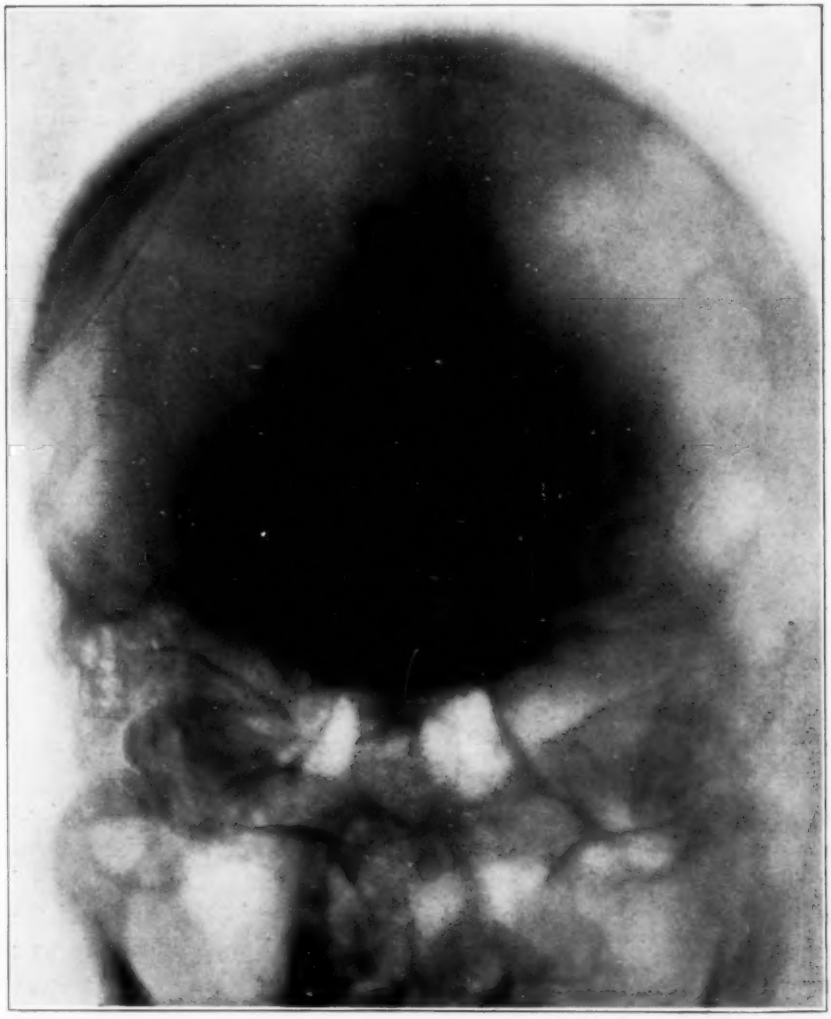

Fig. 4.—Large, single metallic fragment intrahemispheric.

Twenty-four patients gave histories of early hemiplegia which later disappeared entirely or left an insignificant remnant. In contrast, there were sixty patients with definite residual cerebral paralyses. Fourteen gave histories of aphasic disorders of a transitory character, while in sixteen some degree of aphasia persisted as a residual symptom. Four patients described symptoms evidently due to cerebellar

disturbance, all of which recovered entirely. In no case was there evidence of a destructive wound of the cerebellum, a fact to be accounted for by the highly fatal nature of wounds involving the posterior cranial fossa.

Data concerning early sensory symptoms were for the most part unreliable as patients usually fail to note any but perceptual losses, and are even liable to confuse motor paralysis with anesthesia. In ten instances, however, there were fairly consistent histories of superficial anesthesia of unilateral distribution and of temporary duration.

Residual Focal Symptoms.—Under this heading are considered the focal symptoms which persisted while the patients were under observation, in most cases six months or more after the injury.

Motor Symptoms.—Of the entire series of 200, sixty, or 30 per cent., of the patients suffered permanent motor symptoms of cerebral origin. Of these, forty-three were hemiplegic, nine were monoplegic, and eight were paraplegic. The paralysis was of a severe degree in ten hemiplegics and three paraplegics, while in the remaining forty-seven the residual motor disability was comparatively slight when the patients were last examined.

A striking feature of these cases was the marked degree of recovery which invariably occurred. Probably without exception the patients were, immediately following the injury, completely paralyzed in the limbs affected. Twenty of them were admitted to the Cape May Hospital as litter patients; but when last examined they were all ambulatory, and many of them had a very fair amount of function in the paralyzed limbs. Notwithstanding this improvement there remained, in patients having destructive lesions in the motor areas, an irreducible minimum of paralysis.

The residual motor disabilities consisted of disturbances of voluntary motion of the arms and legs, and to a slight degree, of the face. Complete paralysis of a limb was never permanent. The functions of motility most disturbed were those of highly specialized and intricate character. Individual finger movements were uniformly most affected; finger flexion invariably returned in some measure, but extension was weaker and in two cases failed to reappear at all. All movements involving bilaterial groups of muscles were normal or showed insignificant disturbances.

Exaggeration of the tendon reflexes and hypertonicity of the muscles of the affected limbs was the invariable rule, although there was considerable variation in the degree of these conditions. Articular relaxation or increased range of movement in the joints as compared to the normal side was occasionally noted, and was demonstrable

by the greater latitude of movement on passive manipulation after overcoming the hypertonicity of the muscles.

Incoordination constituted a factor in the disability of many of the paralytics, especially those showing a large measure of improvement though actual muscular strength was very fair.

Residual Sensory Symptoms.—Permanent impairment of cutaneous sensory perceptions of touch, pain and temperature was found in only eight cases, and in none was it present as a complete hemianesthesia. On the other hand, thirty patients showed impairment of ability to localize sensory stimuli acurately, to recognize dual contacts, and to appreciate passive movement and position in the extremities. In the

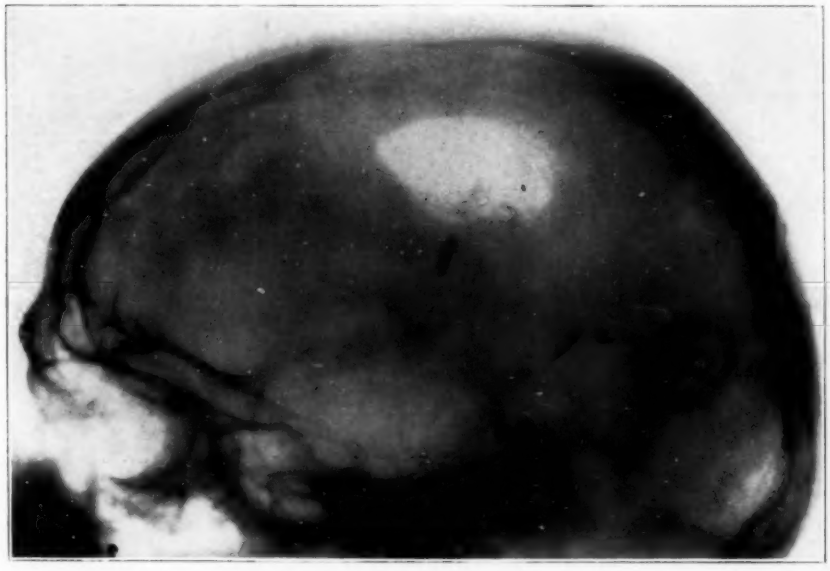

Fig. 5.—Three metallic fragments at a distance from the defect; two bone fragments within the margin of the defect.

same patients there was disturbance of the stereognostic sense. The constant association of impaired sensory discrimination with astereognosis indicates that the latter condition may be considered as a manifestation of the former.

Sensory and motor symptoms were frequently associated in the same case, and those having sensory symptoms almost invariably had motor impairment. The converse was not true, only 50 per cent. of the motor cases having demonstrable sensory symptoms. This relationship of motor and sensory symptoms may in part be explained by the dependence of normal movement, especially its coordination, on the discriminatory element of sensation.

Residual Aphasia.—In sixteen patients disturbances in the use of language remained six months or more after the wounds were received. Of these, ten were of the motor or dysarthric type, three of the sensory type with alexia as the most prominent symptom, and three were of the mixed type, manifesting disturbances both in the expression and in the interpretation of language. In none of the patients were the residual aphasic symptoms of severe degree, and all were able to carry on simple conversations fairly well. The patients with alexia were ultimately able to recognize letters and many words, but did not regain the ability to read understandingly to any practical extent.

Residual Visual Symptoms.—Cerebral wounds were associated with defects in the visual fields in eighteen cases, twelve of which were more or less complete homonymous hemianopsia, three were quadrant anopsias, two were symmetrical paracentral scotomas, and one was almost completely blind. Comparatively slight improvement was noted in the vision of these cases during the period of observation.

Mental Symptoms.—The occurrence of mental disturbance has been mentioned in connection with the general symptoms of cerebral injuries. Aside from the mental symptoms of cerebral concussion and the mild dementias of indeterminate type associated with many brain injuries, a few of the patients have shown late psychic symptoms which evidently resulted from cerebral wounds, and apparently bore some relation to the injured areas of the brain. This was true in four cases in which penetrating wounds involved both hemispheres. In three of these both frontal lobes were affected, and in the fourth a foreign body entered the right frontal region, penetrating to the left posterior parietal region near the cortex. Mental symptoms were pronounced in all of these patients and consisted of disorientation, loss of memory, emotional indifference and disregard of environment and personal appearance. In some measure they resembled the simple dementia of general paresis. Of the many patients with unilateral frontal lesions, some of them extensive, none showed characteristic psychic symptoms. These circumstances indicate the seriousness of bilateral brain lesions, and suggest the theory that either cerebral hemisphere may functionate in a way to minimize the effect of a lesion in the other.

Convulsions.—Convulsions occurred in twenty-eight patients, either before admission or while under observation at Cape May. In four of these the attacks were focal without general involvement, eleven had local spasms initiating general attacks, and in thirteen the convulsions were general so far as observations were recorded, although it is probable that some of these were preceded by unobserved focal

symptoms. Attacks were observed in patients having lesions in the motor area and hemiplegia in which focal signs were definitely absent.

In three of the cases of this group it was found that attacks had occurred prior to military service, leaving twenty-five in which there was evidently a close relationship between the war wounds and the convulsions. In twenty-two of these the wounds involved the parietal region, and in twenty-one there was motor paralysis. In the remaining three cases the wounds were in the frontal, occipital and temporal regions. It is thus apparent that not only focal but general convulsions have been associated with motor areas of the brain in the great majority of the cases, and that irritation of these areas is more productive of general convulsions than of other parts of the cerebrum.

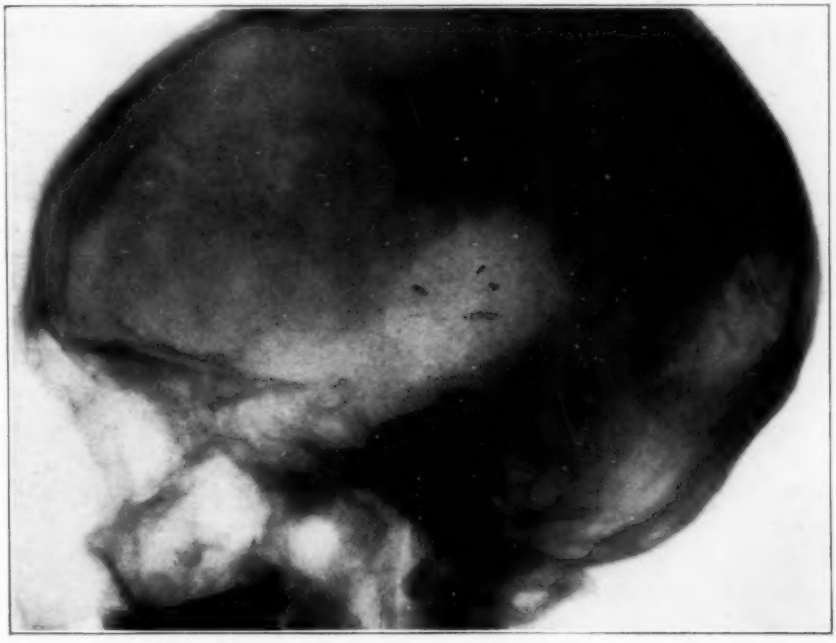

Fig. 6.—One minute bone fragment and three silver clips applied at operation overseas for control of pial hemorrhage.

Eighteen of the twenty-five patients have been free of attacks for several months, and three others have had but a single attack each. Four have had repeated convulsions over a prolonged period, thus evincing a tendency to chronic epilepsy; these were all hemiplegic, and the attacks were of the type which begin as focal convulsions, then become general with loss of consciousness.

The most frequent period for the occurrence of the attacks was soon after the wound had been received or after some operation on

the head. About one half of this group of patients had isolated convulsions at such times without later recurrences.

It should be stated that, as a routine measure, bromids were given in 10 grain doses, three times a day, to all patients having convulsions, and to all those subjected to operations on the head, a measure which no doubt reduced the incidence of the attacks while the patients were under observation.

PATHOLOGY

The degree of injury to the brain varied from insignificant lesions to extensive losses of cerebral tissue. In twenty-three cases intracranial foreign bodies were demonstrated by the roentgen ray, some of them having almost traversed the cranial cavity. Small, indriven fragments of bone were common and were usually located in the vicinity of the cranial defect. In twenty-six cases the wounds were unhealed on admission, most of these having sinuses extending beneath the dura to fragments of dead bone or foreign bodies. One patient, who died four days after admission, had a large temperoparietal abscess and hernia cerebri. This patient was one of the two fatalities in the entire series of head wounds at the Cape May Hospital. The second fatality resulted from a complicating pneumonia and internal hydrocephalus, occurring after the wound had healed and the patient was convalescent.

During cranioplastic operations evidences of cerebral injury were at times noted. In such operations the dura was not usually opened, but occasionally it was necessary, and several times in this way cyst-like cavities filled with cerebrospinal fluid were exposed. In one notable case of this kind the operating surgeon, Major C. C. Coleman, opened such a cavity in the occipital lobe which communicated with the posterior horn of the lateral ventricle.

In estimating the area and extent of the cerebral lesions resulting from war wounds it may be stated as a rule that, in the absence of penetrating foreign bodies, the area of destruction of brain tissue conforms quite closely to the cranial defect, and extends but a few centimeters beneath the cortex. Foreign bodies may penetrate to almost any part of the cerebrum, even traversing the ventricles without causing death. The course of foreign bodies can be estimated by careful roentgen-ray studies, comparing their location with the wound of entrance.

TREATMENT

Besides the surgical treatment which is considered in another part of this report, special courses of treatment were given to practically all of the patients with the object of increasing their general efficiency, and of reducing to a minimum the effect of the disabilities from which

they suffered. School, occupational and workshop courses were prescribed, according to conditions.

Patients with hemiplegia and paraplegia received daily treatments consisting of special massage, passive movements and electricity, also active exercises, employing the affected limbs to a maximum extent on gymnasium apparatus, and in recreational exercises in which handballs and footballs were found to be especially valuable. The results of this treatment were evident in reducing the spasticity and preventing contractures in paralyzed muscles and in procuring a maximum return of function. The training of the unaffected muscles to compensate as far as possible for those of impaired function gave the patients

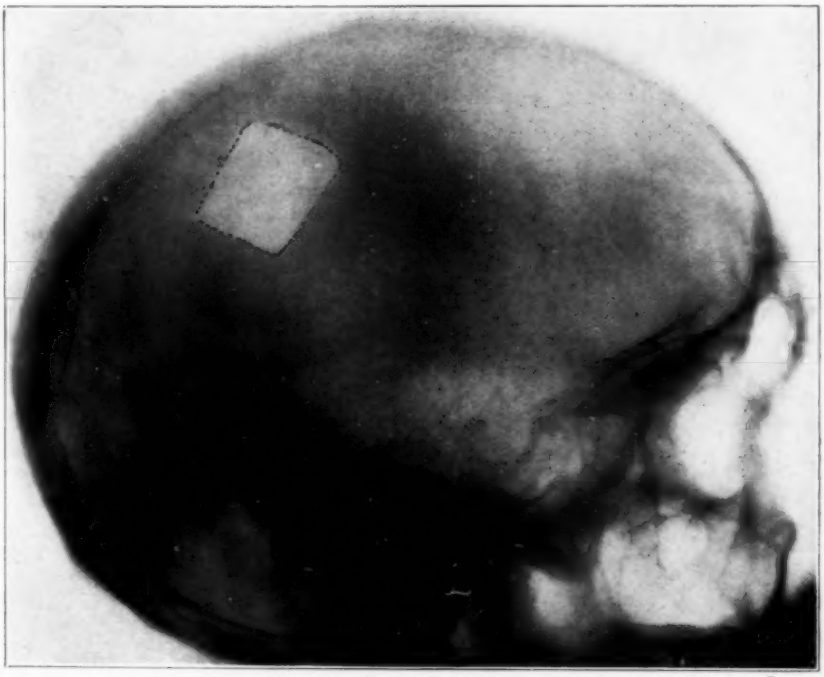

Fig. 7.—Skiagraph of a rectangular defect, in the parieto-occipital region, resulting from removal en bloc of area of skull in the débridement.

greater freedom of action, and the general poise, self-confidence and morale were noticeably improved.

Aphasics constituted another group that received special attention. Trained teachers gave the members of this group daily individual instruction and exercise in conversation, reading and writing adapted to the needs of the patient and the character of his language disturbance. Although we have seen no evidence of the development of new language centers on the normal side of the brain, improvement has been

marked in every patient of this group, the aphasic symptoms of some of whom had previously remained stationary for several months.

SURGICAL CONSIDERATIONS

To the neurologist the most absorbing features of gunshot wounds of the head are the residual disturbances of function, either local or general. The opportunity for an intensive study of the evidences of perverted function in the individual lobes has been taken advantage of by one of us (S. D. I.), and this aspect of the subject has already been reviewed.

The neurosurgical interest in gunshot wounds of the head attaches to those cases only in which an operation is indicated for the relief of one or the other of the following complications: residual abscess, retained foreign bodies, hemorrhage, epilepsy and cranial defects. These complications will be reviewed in the light of our experience at General Hospital No. 11 with 200 head injuries.

1. *Residual Abscess.*—After ten months of observation we have been able to discover but one case of residual abscess, or 0.5 per cent. A patient who died four days after admission, having been operated on on the transport, is excluded from this category. The necropsy revealed an ill-drained abscess in the occipital lobe, a diffuse basilar meningitis and a large collection of pus in the posterior fossa. That the patient should have survived the journey is nothing short of amazing.

The history of the case (Fig. 1) with the residual abscess was briefly as follows:

History.—The patient was admitted to U. S. Army General Hospital No. 11, Nov. 10, 1918, as an ambulatory case. No record accompanied the patient, and he could give no history of his disability. He had received a gunshot wound of the right frontal region near the midline, date unknown. There was a circular cranial defect about 2 by 2.5 cm. without hernia; the roentgen-ray examination showed several bone fragments near the defect and a foreign body 0.5 by 4 cm. in the parietal cortex above and behind the left ear. No definite motor or sensory or other focal symptoms were present. He appeared to understand what he heard, but refused to speak at first. About two weeks after admission he spoke more freely, but was slightly negativistic.

Treatment and Course.—The sudden development of a hernia at the site of the frontal defect was the indication for an exploratory operation. The site of the hernia in the frontal region was explored for abscess with negative results. The patient subsequently died, and the necropsy revealed a chronic meningitis and an abscess cavity along the tract of the missile filled with inspissated pus (Fig. 2). The contents of the cavity were such that any attempt at drainage would have been ineffective. The symptoms, which were complex, and the cause of the death were attributed to a chronic meningitis and secondary hydrocephalus rather than to the abscess.

Report of Necropsy.—Dura: There was an oval opening just to the right of the midline in the frontal portion, through which the brain herniated. Over the inner surface of the anterior half of the dura, covering the left hemisphere, there was evidence of an internal hemorrhagic pachymeningitis.

Hernia: Beginning at a point 3.5 cm. above the base of the brain, anteriorly and just to the right of the midline, there was an irregularly rounded mass, measuring 3 by 3.5 cm. and projecting 1.2 cm. from the frontal lobe.

Ventricles: The right lateral ventricle was greatly dilated, measuring 11.5 by 4 by 6.4 cm. in its greatest dimensions. At one point the left wall of the anterior horn projected 1.4 cm. to the left of the midline. The ependyma lining this cavity showed irregular areas of grayish white thickening. The choroid plexus of the posterior horn was thickened and firmly attached to the right lateral wall, drawing it inward.

The left lateral ventricle was irregularly dilated, measuring 9.2 cm. in its greatest depth with varying length and width. In the superior frontal region, at one point, the cavity approached to within 0.5 cm. of the surface.

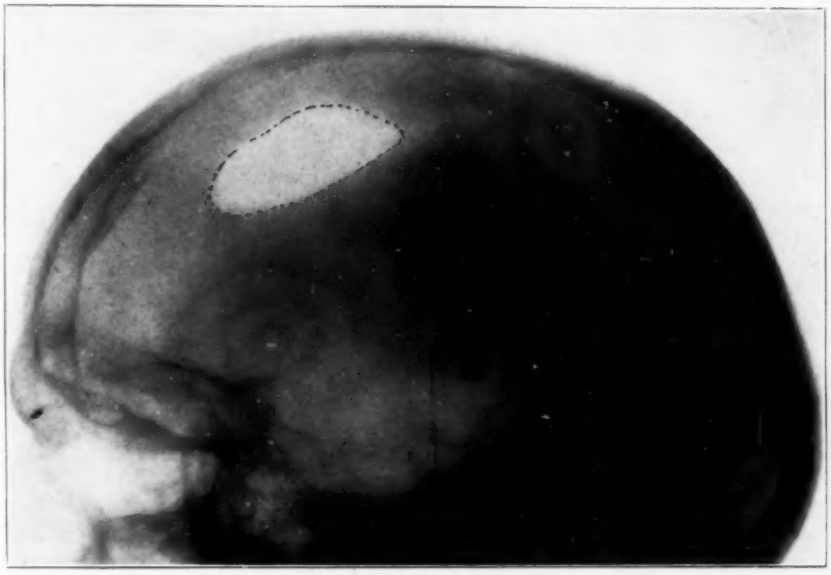

Fig. 8.—Skiagraph of a characteristic oval defect in the frontal region.

Abscess Cavity: Beginning just to the left of the falx, between the frontal lobes and extending outward and backward to the surface in the anterior central gyrus, was a pale orange-yellow granular zone measuring 6 cm. in length and from 1.2 to 2.5 cm. in width, and 3.9 cm. in depth. The central portion of this zone was occupied by a cavity filled with gelatinous and gray amorphous material.

Comparing our figures with those of other countries, we find that, whereas our percentage of residual abscess was only 0.5 per cent., Sargeant and Holmes reported thirty-seven in a series of 1,567 cases, or 1.3 per cent., nearly three times as many. The difference between

the two series may be due to the fact that a much larger proportion of cases was evacuated to England after a shorter interval, sometimes ten days or two weeks, whereas with the A. E. F. the interval was longer, and in this interval some cases of residual abscess may have been operated on before transport. I have excluded from consideration fifteen cases of superficial abscesses which were complications of surface infection and osteomyelitis.

Retained Foreign Bodies.—Retained foreign bodies, strictly intracerebral, were observed in 10 per cent. of our series. The fragments, either bone or missile, were of various sizes, single or multiple, superficial or deep (Figs. 3, 4, 5 and 6). In no instance, save two, were they the cause of symptoms. One of these cases has already been described as a case of residual abscess; the other was associaated with a jacksonian epilepsy with the following history:

History.—The patient, who was struck by a high explosive shell in the frontal region just above the eyebrow, received first aid at once. He was operated on at the field hospital; fragments of bone were removed and the wound closed with tube drainage. Sept. 27, 1918, the wounds had healed.

He was operated on June 9, 1919, at General Hospital No. 11. Craniotomy was performed for removal of a foreign body which was localized by measurements and identified by its relation to the bifurcation of the anterior middle meningeal artery, grooves of which could be seen in the skiagraph. The foreign body was located with a needle and extracted with a magnet. It appeared to be covered with soil. The sinus from which it was extracted was disinfected with dichloramin-T and the wound closed. Just before leaving the operating table the patient had a short convulsive seizure involving left face, left arm and left leg.

The foreign body was readily localized and its close proximity to the precentral convolution justified us in the belief that it might have been the exciting factor in the epileptic seizures. At all events this possible relationship, the fact that there had been but two seizures, that the foreign body could be removed without risk to life or harmful damage to the brain, justified its removal.

Our percentage of retained foreign bodies, 10 per cent., is almost identical with that recorded by Sargeant and Holmes; 164 in 1,567 cases, or 11 per cent. That the percentage is so low speaks for the thoroughness and the success with which the original operations were performed at the field and evacuation hospitals. The percentage of fatalities, as given in the British table, was, however, higher than ours. Of their 164 cases, twelve, or 7 per cent., died, whether with or without operation is not stated; of our twenty-three cases, one, or 4 per cent., died.

The indications for operation in the presence of foreign bodies are clearly defined and may be summed up as follows: Those causing encephalitis or epileptic seizures should be extracted and those appar-

ently latent should be left alone. At least these are the principles which have governed us in our selection of cases for operation. I have purposely excluded from our statistics of foreign bodies minute sequestrums or fragments which have been found in sinuses or infected wounds leading to or in the cortex of the brain. There were, all told, seventeen of these, revealed by roentgen ray, and removed merely as a phase of the wound treatment.

Hemorrhage.—In the report of Sargent or Holmes, of the forty-six out of 1,239 cases dying after evacuation to England, eleven were

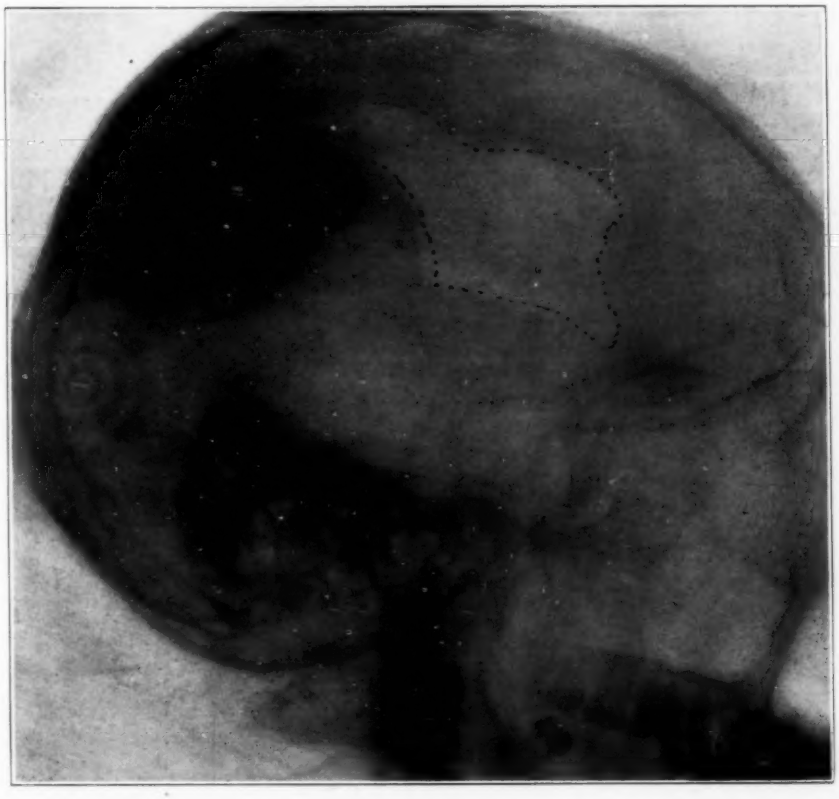

Fig. 9.—Skiagraph of an irregular defect in the parietal region.

attributed to intracranial hemorrhage. This complication has not been observed in our series.

Epilepsy.—Whether epilepsy will be admitted as a surgical complication we leave to the reader's decision. There have been so many ill-advised operations for epilepsy on the part of surgeons, that we submit the question with due apologies for their shortcomings, and yet the frequency of epilepsy is such and the resulting disability

so great if unrelieved, that the subject cannot be lightly passed over. This complication from its general neurological aspect has already been dealt with, but I will quote a few statistics merely to emphasize its importance.

At the Third Interallied Congress held in Val-de-Grace, Nov. 5, 1917, Tuffier and Guillain reported 676 cases, or 10.14 per cent., in 4,262 cases and in the publication of the Medical Research Committee of Great Britain, Adie and Wagstaffe reported thirty-seven out of 610 cases, or 6 per cent. In our 200 cases there have been twenty-five, or 12.5 per cent. These percentages correspond very closely, but in literature generally one finds an extraordinary variation in the quotations. For example, Villande[1] makes the unbelievable statement that of 450 cranial wounds, in 303, or 70 per cent., secondary operations were performed for which the sole indication was the development of epileptic seizures. Our policy in the matter of operative interference has been briefly this: We have not recommended operation in any case as a preventive measure; we disapprove operation, except in those cases in which the presence of a foreign body or a cranial defect and the resulting dural adhesions might be regarded as responsible for the seizures. Under these restrictions, we have operated on four cases.

CASE 1.—The patient, who was admitted on a litter Sept. 25, 1918, was wounded July 18, 1918. He had an infected gunshot wound of the left parietal region with a defect 4 by 5 cm.; hernia cerebri was present. He had a convulsion after admission and curettage of wound Oct. 19, 1918, with a second curettage April 10, 1919. The wound finally healed about May 1, 1919. He has had no convulsion since January and only one after operation.

CASE 2.—The patient, who was admitted Dec. 21, 1918, received July 13, 1918, on the Italian front, a gunshot wound in the midfrontal region with a small defect 1 by 1 cm. The wound had healed. The roentgen ray showed a foreign body in the right precentral area about 1 cm. from the dura. The patient showed no paralysis. While on furlough in April he had a general convulsion coming on during sleep, and in May had local jerking of the left arm without loss of consciousness. June 9, 1919, the foreign body was removed. He had three convulsions on the day of the operation but has had none since.

CASE 3.—The patient, who was admitted Dec. 12, 1918, received July 4, 1918, a gunshot wound of the right parietal region with a defect 4 by 7 cm. He gave a history of convulsions before admission and had two convulsions December 20. He developed status epilepticus Jan. 20, 1919. Cranioplasty was performed Jan. 21, 1919. He has had no convulsion since.

CASE 4.—The patient was admitted Aug. 30, 1918, having received a gunshot wound of the left parietal region and lost his left eye on May 13, 1918. The parietal defect was 4 by 4 cm. He had convulsions soon after admission. Cranioplasty and cartilage graft were performed Oct. 24, 1918. He had one focal convulsion after operation, but has had none since.

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1. Villandre, C.: Healing of Skull Wounds, Arch. de med. et pharm. mil. 68:546 (Oct.) 1917.

Late Cerebral Hernia.—It was a source of surprise that we received no cases of cerebral hernia, despite the fact that there were twenty-six cases of open wounds and 153 of cranial defects. Again, to quote statistics, Tuffier and Guillain report 54 in 6,664 cases of gunshot wounds, 0.81 per cent.; in only three of 656, 0.4 per cent., cases

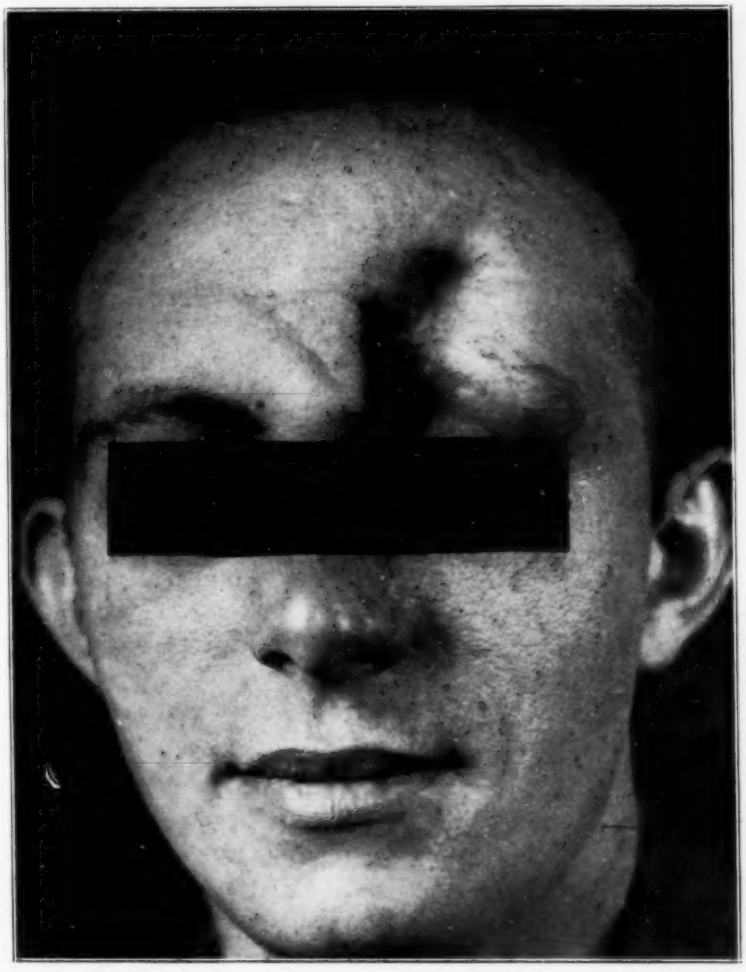

Fig. 10.—Characteristic defect in the frontal region.

evacuated to England, was the hernia larger than a golf ball (Adie and Wagstaffe); in ninety of 1,567 cases, 5 per cent., evacuated to the hospitals of Great Britain, were there large cerebral hernias (Sargeant and Holmes). Needless to say, we would not have included late cerebral hernia among the indications for surgical intervention.

Cranial Defects.—With few exceptions—one residual abscess, one intracerebral foreign body and four epilepsies—we have been confronted with no condition justifying resort to operation other than the presence of cranial defects and of these there were, all told, 153 out of 200 cases, or 70 per cent. Of the number, twenty-six were from 1 to 4 cm. in diameter, fifty-one were from 4 to 16 cm. in diameter, thirty-seven were 16 cm. and over in diameter.

The shapes of the defects were round, irregular or rectangular. In all cases the defect followed the deliberate removal of bone as a part of the preliminary débridement; in some instances, fragmentarily; in others, evidently en bloc (Figs. 7, 8 and 9). There was a noticeable difference in the physical condition of the tissues of the integument and underlying it. In some instances there was marked pulsation; in others, not; in some, when the patient stooped, the underlying brain would protrude beyond the level of the scalp; in some the integument contained dense connective tissue which served as adequate protection to the brain; in others the integument consisted solely of a thin reddish cicatrix which often for lack of nutrition exhibited a superficial ulceration. We assumed that in the latter instance there was no dura mater at the site of the defect, that originally there had been a hernia cerebri and, in the process of wound repair, the brain was first covered with a thin layer of granulation tissue, which ultimately cicatrized.

There has been much discussion as to the propriety of repairing cranial defects, even though the operation is, in proper hands, unattended with risk and uniformly successful. First, we will enumerate the conditions under which the operation is not recommended:

1. For the repair of small defects.

2. For the repair of defects in which the brain is adequataely protected by a dense cicatrization and there is no protrusion when the patient stoops.

3. For the treatment of cerebral hernia.

4. For the relief of epilepsy except under the conditions previously noted.

5. When there are any grounds of suspecting an abscess or when there are foreign bodies which may require removal.

The cranial defects should not be repaired, according to Guillain, if the cerebrospinal fluid is not absolutely normal as to tension, chlorid and albumin contents; if there is a pupillary stasis or a choked disk; if there have been partial or general epileptic phenomena; if there are signs of serious meningeal symptoms.

Under what circumstances then is an operation indicated? These may be briefly summarized as follows:

1. For cosmetic reasons, especially when the defect is in the frontal region below the hair line. There are many of these.

2. In certain selected cases of epilepsy.

3. In the presence of a large defect where the brain is exposed to trauma.

4. When the patient is apprehensive, because of the defect and he dreads the possibility of a direct blow on the uncovered cortex.

5. In a few cases where the symptoms are wholly subjective, an operation is advisable in the hope that, combined with the influence of suggestion, the neurosis will be relieved.

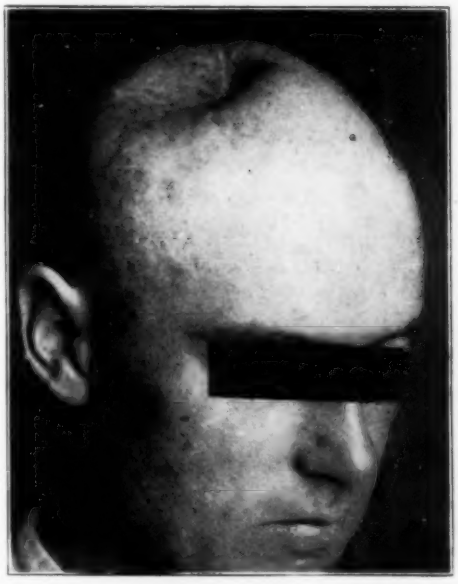

Fig. 11.—Characteristic defect in the parietal region.

Under these indications we have operated on fifty-nine out of 153 cranial defects, or nearly 40 per cent.

There are some who not only do not advise the operation under any circumstances, but who believe operation is distinctly harmful. Dide and Claude thought it psychologically obnoxious, to be a factor in the persistence of subjective symptoms and in producing a neurasthenic syndrome. Andre Thomas said that sometimes epilepsy developed after operation, or if present at the time, was aggravated by it.

We have not included in the indications for cranioplasty the possibility of affording relief to a miscellaneous group of symptoms of

which patients often complain who have sustained an intracranial injury. I refer particularly to headache, vertigo, tinnitus, drowsiness, lack of initiative, depression, amnesia and occasionally vomiting. It may be said, however, that not infrequently after an operation for the repair of the defect, there is a manifest improvement in the patient's condition. One of the patients said he had not felt so well since the day he was injured. The headache is less constant, the patient, hitherto depressed, is brighter and more optimistic, he complains less of vertigo, especially on stooping, changing position or taking exercise. I venture on no explanation for this temperamental change, but merely record it as a phenomenon which I had observed previously in the intracranial injuries of industrial accidents and more recently in the gunshot wounds of the head.

Cranioplasty.—Since the outbreak of the war there have been numerous contributions to the subject of cranioplasty. This is quite natural since the opportunity for performing this operation has increased in untold numbers. Many an author writes as though the surgical repair of defects had not been practiced before the war. As a matter of fact, the technic of the operation had been elaborated in every detail and no revision was necessary in its application to the defects due to gunshot wounds. It has been my practice for many years to repair cranial defects with a bone transplant, composed of the pericranium and a thin shell from the outer table of the skull. The operation is devoid of risk, easy of performance and uniformly successful. But in the war literature one finds many other methods recommended, notably the use of cartilaginous grafts and skull fragments taken from the dead. French writers spoke so enthusiastically about their results with grafts of costal cartilage that I was tempted to substitute these for the external table graft which I had been in the habit of using. The immediate results were not as satisfactory in that the cartilage graft collapsed in the wound, and left a shallow saucer-like depression, and of the late results there have been reported instances in which the graft was absorbed. After a brief trial of the cartilage graft I soon abandoned it and resumed my original prewar technic.

A COMPARISON OF THE ANTERIOR HORN CELLS IN THE NORMAL SPINAL CORD AND AFTER AMPUTATION *

A. E. TAFT, M.D.

Formerly Curator, Department of Neuropathology, Harvard Medical School; Assistant Physician, Pennsylvania Hospital for Nervous and Mental Diseases

PHILADELPHIA

This work arose from the negative results of an examination, by a student in the department of neuropathology, of three spinal cords from cases having had amputation of a limb sometime previous to death. The findings were so little in accord with the idea of postamputation pathology gained from various textbooks, that a further examination of cords was undertaken in the same manner, from subjects not showing signs of cord lesions during life, and from that point of view called normal. Except for this general precaution material was chosen at random.

A variety of findings are recorded in the literature as a result of investigations of the changes in the anterior horn cells following amputation. No attempt has been made to include a complete literary review; the plan is only to give an idea of the varied results arrived at by a number of experienced investigators.

LITERATURE

Campbell[1] says, after considerable work on this subject, "it may be explained that in consequence of such a lesion alterations occur in the spinal cord and these in course of time occasion striking and characteristic appearances. In long-standing cases the predominant change is a homolateral atrophy, represented by a general reduction in volume of white and gray matter alike, and involving those particular segments of the cord which receive and give off the sensory and motor nerves which originally supplied the skin and muscles of the amputated member. Wasting of the gray substance is accompanied by the numerical reduction of its contained nerve cells, both large and small, and while

* Contribution in a series offered to Professor E. E. Southard in honor of the decennium of the Bullard Professorship of Neuropathology, Harvard Medical School.

1. Campbell, A. W.: Changes in the Spinal Cord Following Amputation, British M. J., March 14, 1896, p. 663; Post-Amputation Change in the Cerebral Cortex. Histological Studies on Localization of Cerebral Function, p. 47.

all the cell collections in the anterior cornu suffer, one special group may be singled out as being specially prone to atrophy, namely, the posterolateral."

Marinesco,[2] one of the earlier investigators of this question, is one very often quoted. His report includes three cases: one with duration of twenty-one years; one ten years, and the third of unknown duration. He found changes in anterior horn cells; in the first two cases a reduction in number on the side of the amputation, not only in the lateral, but in other groups; in the third case neither the cell count in the posterolateral group, nor of all cells in all groups have sufficient significance to conclude that there is any definite change in the cells.

Edinger[3] reports a case of prenatal amputation of the left lower arm with death at 52 years of age. The left side of the cord corresponding to the four lower cervical and first two dorsal segments showed a distinct atrophy of the anterior horn, which was greatest at the level of the sixth and seventh cervical segments. There was also a distinct decrease in the size and number of the anterior horn cells, especially at the anterolateral and posterolateral angles of the anterior horn. In three additional cases of postamputation, no convincing proof of cell atrophy was found in the anterior horns.

Van Gehuchten[4] examined the lumbosacral cord from a case twelve years after amputation of the lower extremity in the middle portion of the thigh. The anterior horn cells representing the foot muscles were normal, while the cells representing the muscles below the knee had disappeared, in spite of the fact that the axons of both groups of cells had been divided.

Déjerine and Mayer[5] studied eight cases of amputation varying from four to forty years following amputation. They found in all cases a definite decrease in the size of the side of the cord corresponding to the amputation, affecting both white and gray matter, both anterior and posterior. In only one case were histologic changes found; this was following amputation of the thigh, of thirty years' duration. In the middle of the lumbar enlargement a definite decrease in the number of anterior horn cells appeared, the number being only a third of the normal side, and the anteromesial group was the one most affected.

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2. Marinesco, G.: Ueber Veränderungen der Nerven und des Rückenmarks nach Amputationen; ein Beitrag zur Nerventrophik, Neurol. Centralbl. **11**:463, 1892.

3. Edinger, L.: Rückenmark und Gehirn in einem Falle von angeborenem Mangel eines Vorderarms, Virchow's Archiv. **89**:46, 1882.

4. Van Gehuchten: Handbuch der Anatomie des Nervensystems, **1**:139; Bull. de l'Acad. roy. de Belgique, 1898, p. 222; Presse méd. Jan. 4, 1899.

5. Déjerine and Mayer: Bull. Soc. de Biol., 1878.

Elders,[6] in an excellent article, reports the examination of the cord of a person born without the left lower arm. Death occurred at 54 years of age. In the lower half of the left cervical segment the postero-lateral angle was rounder and shorter than the right and contained fewer cells.

Dreschfeld[7] examined a case of amputation of the left thigh fifteen years after operation, and found atrophy of the anterior horn and some change in the posterior horn in the lower part of the left lumbar enlargement, with decrease in number and atrophy of the nerve cells of the intermediolateral group.

Kahler and Pick[8] report two cases, one of which was an amputation of the lower third of the left thigh, eighteen years after operation. The anterior horn cells were decreased in the anterior group. The second case was an amputation of the left forearm in the lower third. He reports partial atrophy and decrease in the number of cells in the lateral group.

Dickinson[9] reports findings on a case of amputation of the right leg fifteen years before death, in which there was a decrease in the number and size of the nerve cells of the corresponding side.

Genzmer[10] found decrease in size of anterior horn and in the number of anterior horn cells on the amputated side in a case of amputation of the lower third of the right thigh 30 years before death.

Hayem[11] examined the cord in the case of exarticulation of the wrist, five years after operation, and found general atrophy of the anterior horn with atrophy of nerve cells, most marked at the level of the eighth cervical and first dorsal nerves.

Friedreich's[12] case of amputation of the left forearm twelve years before death showed no changes in the cord; the anterior horn cells were intact.

ANIMAL EXPERIMENT

Experimental work also has been done on various laboratory animals, both by section of nerve trunks and by amputation of extremities.

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6. Elders: Monatsch. f. Psychiat. u. Neurol., 1910.
7. Dreschfeld: On the Changes in the Spinal Cord After Amputation of Limbs, J. Anat. & Physiol. **14**:424, 1879.
8. Kahler and Pick: Weitere Beiträge zur Pathologie und Pathologischem Anatomie der Central Nervensystems, Arch. f. Psychiat. **10**:360.
9. Dickinson: On the Changes Which Occur in the Spinal Cord After Amputation of a Limb, Compared with the Changes Found in Progressive Muscular Atrophy, Trans. of Path. Soc. of London, **24**: 1873.
10. Genzmer: Veränderungen im Rückenmark eines Amputierten, Virchow's Archiv. **66**:265, 1876.
11. Hayem: Arch. de Physiol. Norm. et Path., T. V., 1873, p. 504.
12. Friedreich: Ueber Progressive Muskelatrophie, Berlin, 1873.

Van Gehuchten[4] divided the sciatic nerve and later found only the cells of the posterolateral group affected.

Homén's[13] work on animals showed a slight atrophy and decrease in the number of cells of the anterior horn. The individual cells were found in general somewhat smaller than the corresponding ones on the opposite side. No changes were found in the brain.

The experimental work of Warrington[14] is a more recent and detailed investigation of the changes of the anterior horn cells after section of the spinal nerve roots. In some instances changes were found both in anterior and posterior horn cells corresponding to the side operated on. He reports finding changes not only on the corresponding side, but also on the opposite side. "After section of several posterior roots from the VIth to the IXth post thoracic inclusive, a considerable percentage of obviously altered cells are found. Their distribution in the case of the cat is practically limited to the VIIth and VIIIth segments, and especially to the postero-lateral group of cells in those segments. In the monkey the upper part of the VIIth segment is picked out. The effect is to a very slight extent a crossed one and presents the remarkable feature that more affected cells were found in the VIth segment of the crossed side than on the side of the lesion." Further, "the view I wish to maintain is that the changes are the result of the withdrawal of the afferent impulses which normally impinge on cornual cells. Histological evidence shows that the postero-external group of anterior horn cells is most richly innervated by the collaterals from the posterior roots." He thus explains the degeneration of this group of cells, and adds further with emphasis that the changes occurring after section of the posterior roots are much more intense and that the ultimate destruction of these cells is more likely to occur than after the division of the anterior roots alone. After section of both anterior and posterior roots all the larger cells on the side of the lesion showed changes. The greater number were only slightly affected compared to the extent seen after section of an anterior nerve root alone; others, however, showed a much more marked chromatolysis, the changes resembling those found after section of the afferent roots. He finally concludes that there is a tendency for the altered cells to return to their normal structure regardless of the regeneration of their axons, in conformity with the opinions of Van Gehuchten and Nissl.[15]

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13. Homén: Veränderungen des Nervensystems nach Amputationen in Ziegler: Beiträge zur Pathologischen Anatomie, 1890, p. 304.

14. Warrington: Observations on the Structural Alterations Observed in Nerve Cells, J. Physiol. **23**: 1898.

15. Nissl, F.: München. med. Wchnschr., 1898, p. 6.

FROM TEXTBOOKS

Concerning the change in the anterior horn cells following division of their fibers Oppenheim[16] says: "The Nissl stain shows destruction of the granules and an excentric position of the nucleus. If a restitution in the periphery takes place, regeneration results. If, however, there is no peripheral regeneration, further change in the form of atrophy takes place in the cells."

Van Gehuchten: The section of the axon leads to a rapid swelling of the cell protoplasm, and consequent displacement of the nucleus. He believes that in a small number of cases the swelling of the cell body takes place so rapidly and the force which pushes the nucleus to one side is so powerful that it is driven completely out of the cell body. These cells are the only ones, he concludes, which completely atrophy, while all the others slowly return to normal.

Déjerine and Thomas:[17] For localization studies, division of nerves has been made. In man, the cords of persons having had amputations have been studied with the same end in view, but the application of this method has not always been free from reproach.

Schäfer: If the degeneration has been caused by section of the axon, the reparative process is very slow, so that it may be three or four months before it is completed. At the end of this time the nerve-cell bodies have resumed their original appearance even though reparation of the cut nerve may be incomplete.

Barker:[18] The motor fibers of the central stump gradually diminish in number; in some instances they appear to vanish almost totally, and a large number of the motor cells of the ventral horns dwindle in size and after a time actually may be lost.

MATERIAL EXAMINED

The tissue was prepared in serial paraffin sections, 6 microns thick, and stained by the cresylechtviolet cell-stain method.

Material from three cases of amputation was examined as a starting point of this work, and mainly from the point of view of loss of anterior horn cells. Sections were examined from the segments of the cord corresponding to the part amputated, and the cells of both anterior horns were counted.

Case 1.—Man, aged 79, sixty years after amputation of left arm. The anterior horn cells showed no chromatolysis, but many of the cells were well filled with lipochrome. This is not unusual in view of the age of the man. One hundred and thirty-three serial sections were counted. The variation in individual sections was considerable, but the final average of all counts was, right 17, left 19, or an average of two cells more on the amputated than on the opposite side.

In the brain the Betz cells and other large elements contained much lipochrome, but showed no reaction to the amputation.

Case 2.—Man, aged 65 years, recent amputation of lower third of the left leg. The anterior horn cells were very little altered. There was no chromat-

16. Oppenheim, H.: Lehrbuch der Nervenkrankheiten, **1**:148.

17. Déjerine and Thomas: La Moelle Epiniere, 165.

18. Barker, L. F.: The Nervous System, New York, p. 230.

olysis, and very little lipochrome. Forty-one serial sections were counted. Variation in individual sections was as much as 62:38 between the two sides. The average of the total number counted was, right 52, left 55, or an average of three more cells on the amputated side. As the operation in this case was performed only the day before death (following gangrene of the foot), no permanent change in nerve cells would be expected. No axonal change had taken place.

Case 3.—Female, aged 73, old amputation of right thigh. The anterior horn cells contained a great deal of lipochrome, but also some normal Nissl granules. A total of 41 counts was made on serial sections. The variation in individual sections was as great as 13:32. The total average was, right, 21.8; left, 23.9, or an average of two more cells on the nonamputated than on the amputated side.

In addition to these counts others were made on material from a variety of cases, with the single precaution that they show no clinical signs of cord involvement. Both sexes were included, and the ages ranged from 11 months to 71 years.

One case considered particularly suitable for use as a normal control was that of a young male adult, 37 years of age. Here the average of 67 counts in the cervical region was 50 on one side and 54 on the other; of 53 counts in the dorsal region, 12.0 on one side and 12.9 on the other; in the lumbar region the average of 62 counts was 49.5 on one side and 52.0 on the other. This variation is seen to be greater than in the cases of amputation. In Marinesco's[2] work the greatest number of sections of one level counted is sixteen, and the variation in the sum total is 245:223. In the instances where a smaller number of sections were counted, the difference between the two sides was even greater. In the case of the normal control just described, in one group of 21 counts, the total was 938 on one side and 1,181 on the other; but in the final sum of all counts, 67 in all, the number was 2,603 on one side and 2,684 on the other.

In the accompanying table the detail of the entire number of cases counted may be seen. No attempt was made to identify the exact segment of the cord from which the blocks were taken, as much of the material was already embedded without regard to this detail; consequently, there is a great difference in the count at the varying levels. It may be noted also that numbers are much larger up to and including Case 26 than in the subsequent ones. This is due to the fact that in the earlier cases all cells were counted which represented the full contour of a cell including any of its processes, exclusive of whether it contained a nucleus or not. Later, only those cells were counted which contained a stained nucleus. But since the comparison is that of the two sides of the cord, and not between different specimens, the relation is not altered.

TABLE 1.—COUNTS OF ANTERIOR HORN CELLS IN SERIAL SECTIONS OF SPINAL CORD

Case	Diagnosis	Sex	Age	Following Amputation						Sections Counted
				Cervical		Dorsal		Lumbar		
				1	2	1	2	1	2	
1	Amputation, left arm	M	79	19.3	17.4	....	....	....	....	133
2	Amputation, left leg	M	65	....	....	....	....	55	52	41
3	Amputation, right leg	F	73	....	....	....	....	23.9	21.8	41
				Without Amputation						
4	Congenital syphilis	F	13	15	16	6	5.7	16.–	17.8	C.15–D.17–L.18
5	Not insane	M	6	14.–	18.6	4.9	5.–	18.–*	15.–	C.15–D.15–L.16
6	Organic dementia	M	42	23.–	31.8	5.5*	6.–	37.5	28.–	C.16–D.14–L.15
7	Mongolian idiot	F	6	11.8	12.–	11.6	14.–	28.8*	27.–	C.15–D.26–L.26
8	Idiot	M	17	43.–	52.7	10.7	10.8	29.4	31.–	C.18–D.22–L.19
9	Imbecile	F	46	36.–	32.–	6.–	6.–	34.–	31.–	C.18–D.17–L.15
10	Alcoholic	F	38	30.7*	28.–	6.–*	6.–	33.–*	37.–	C.16–D.17–L.15
11	Auto-intoxication	F	42	29.–*	34.–	7.–*	6.–	33.7*	35.–	C.16–D.34–L.18
12	Alcoholic delirium	F	32	27.6*	41.6	6.8*	7.–	27.–	28.6	C.15–D.33–L.18
13	Special control	M	37	50.4	54.3	12.–	12.9	49.5	52.4	C.67–D.53–L.62
14	Presenile depression	F	68	23.5*	22.5	4.–*	5.–	19.9*	17.9	C.15–D.15–L.15
15	Unclassified	M	71	19.4	19.6	2.8	3.8	19.–	17.8	C.15–D.15–L.15
16	Delirium	F	..	14.9	13.4	2.6	2.5	17.8	19.2	C.15–D.36–L.15
17	Not insane	F	51	....	....	8.–	8.6	22.7	21.8	D.34–L.14
18	Dementia praecox	F	40	21.–*	25.–	5.9*	5.4	14.–*	11.–	C.21–D.18–L.22
19	Imbecile	M	34	34.3*	21.3	11.3*	6.3	24.9*	23.4	C.20–D.25–L.20
20	General paresis	M	39	20.4*	20.2	3.–*	3.1	8.2*	8.5	C.20–D.20–L.20
21	Unclassified	M	21	....	....*	4.–	4.–	14.–*	13.5	D.41–L.25
22	Chronic dementia	F	40	8.9*	8.1	1.8*	3.–	11.–*	9.1	C.31–D.21–L.33
23	Puerperal mania	F	24	6.6*	7.8	2.1*	2.–	7.4*	8.7	C.21–D.22–L.20
24	Uremic intoxication	F	28	4.–*	5.6	1.3*	1.9	7.7*	6.6	C.23–D.32–L.21
25	Dementia praecox	F	35	6.5*	6.9	1.2*	1.2	10.3*	10.4	C.20–D.22–L.20
26	Imbecile	M	36	5.5*	3.4	2.1*	2.2	....	....	C.16–D.17
27	(Infant)	..	11 mo	6.9*	6.2	3.–*	3.8	13.–*	12.6	C.23–D.22–L.22
28	Unclassified	F	3 yr. 10 mo	....	....	3.–*	2.7	17.–*	19.–	D.24–L.24

* Left side of cord.

These results contribute something also to the question of whether the number of cells is greater on one side or the other from the standpoint of right and left handedness. It will be seen that there is no constant relation throughout the list, the number being greater as an average on one side in some instances, and less on the same side in another, with very slight variation in any case, and no more in the cervical than in the lumbar region. By means of a low power lens, it may be seen in progressing from one section to another that one horn contains the greater number of cells, first on one side and then on the other, and the same variation is seen in the individual cell groups.

It may be noted here that Bruce[19] found only the fifth cord specimen examined suitable for use in preparing his Atlas of the Spinal Cord.

SUMMARY

Cell counts were made on the anterior horns of the spinal cord in twenty-eight cases. Three of these were cases following amputation of an extremity. The twenty-five additional cases were said not to have had signs of a cord lesion.

19. Bruce, A.: Atlas of the Spinal Cord, 1901, p. 3.

Variations in the counts between the two sides in the amputated cases was not more than two or three cells in the final average. In two of these the greater number was on the side corresponding to the amputation.

Variations in the counts between the two sides in the cases without amputation was at times greater than with amputation.

In the sections where identification was made of the right and left sides, there appears no uniform difference between the two corresponding counts.

CONCLUSIONS

The literature contains a variety of opinions concerning the permanent changes in the anterior horn cells of the spinal cord following amputation.

A fairly large number of counts of anterior horn cells of the spinal cord from cases without amputation show as great a variation in the number of cells as in cases with amputation.

In the material examined there seems to be no constant variation between the number of anterior horn cells in the right and left sides of the spinal cord.

NONCONCOMITANCE OF SPINAL FLUID TESTS*

H. C. SOLOMON, M.D.
BOSTON

TESTS APPLIED TO SPINAL FLUID IN DIAGNOSIS OF DISEASE OF CENTRAL NERVOUS SYSTEM

There are five laboratory tests commonly applied to the spinal fluid in the diagnosis of disease of the central nervous system, namely: (1) Wassermann reaction; (2) test for globulin; (3) test for increased albumin; (4) cell count (increase called pleocytosis); (5) Lange's colloidal gold test. A positive Wassermann reaction in the spinal fluid is practically specific for neurosyphilis. A positive result in the other tests, in a general way, proves a pathologic reaction of an inflammatory nature in the central nervous system. At any rate, where an inflammatory condition of the nervous system exists tests 2, 3, 4, and 5 are usually positive. In other words, in meningitis, encephalitis, tumor with meningitis sympathica, vascular insults with secondary inflammatory reaction, traumatic injury, multiple sclerosis, these four tests are generally positive. In paretic or tabetic neurosyphilis all five are usually positive.

The subject of this paper is the consideration of the relation of these tests one to the other, the identity or nonidentity of one to the other and their independent appearance and disappearance in disease conditions. There are three methods of study applicable to this problem: (*a*) chemical and biologic analysis of the substances, (*b*) review of the spinal fluids obtained from a large series of cases with attention to anomalous or unusual relations or nonrelations of findings, (*c*) frequent examination of fluids from cases of neurosyphilis undergoing treatment and in which the spinal fluid findings are influenced in the process of improvement.

WASSERMANN TEST

The Wassermann reaction in the spinal fluid is a biologic reaction pathognomonic of syphilis of the nervous system. As a rule when present it indicates an inflammatory change in the nervous system, but occasionally it is positive when the involvement is chiefly vascular, and not inflammatory. The so-called Wassermann bodies, whatever that substance is which produces deviation of complement, is an

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*From the Massachusetts State Psychiatric Institute, Boston.

*A paper presented to Dr. E. E. Southard, in honor of the decennial of the Bullard Professorship of Neuropathology at the Harvard Medical School.

unknown element. There is some evidence to show that it is related to the albuminous content of the blood serum and by analogy, therefore, to the albuminous content of the spinal fluid. Thus Bruck has shown that an albumin reaction occurs in many cases giving a positive Wassermann reaction which is different from that obtained in Wassermann negative cases. Weston showed that the Wassermann producing substance does not pass through a dialyzing thimble which is impermeable to albumin. In some unpublished work at our laboratory we have shown that the addition of pure globulin to a spinal fluid giving a negative Wassermann reaction, will cause the reaction to become positive. However, these bits of evidence are far from conclusive that the Wassermann producing body is contained in the albumin and there is evidence, such as that brought forth by McDonagh, that it is a lipoid element with which the Wassermann reaction is associated. The evidence we bring in the body of this paper is against the relation of the Wassermann reaction to globulin or albumin.

It is of course evident that there is no relation between the Wassermann reaction and pleocytosis. Pleocytosis is evidence of inflammation, the Wassermann reaction of syphilis. Even where the inflammation is of syphilitc origin, the Wassermann reaction may be negative in the presence of a pleocytosis or, what is much more common, the pleocytosis may be absent with a positive Wassermann.

TEST FOR GLOBULIN

Globulin by definition is an albumin which is salted out of solution by half saturation with ammonium sulphate. Globulin does not occur normally in the spinal fluid. When present it is an evidence of pathology, and it is usually considered as proof of an inflammatory type of reaction. It also may be present, however, when there has been seepage of blood into the spinal fluid through a hemorrhage or vessel changes. If globulin is present in the spinal fluid, theoretically it means that there is an increase in the total amount of albumin, as this is an addition of an albuminous substance to the albumin normally present. As albumin which is precipitated by an acid such as trichloracetic, is normally present in the spinal fluid in small amounts, it is increased in amount whenever globulin is present. However, it is not dependent for its increase solely on globulin, but is increased as a result of inflammation. While we have mentioned that increased globulin cannot exist without a theoretic increase in the amount of albumin, albumin can be present in increased amount without the presence of globulin. We will later show that this actually occurs. While again the globulin, albumin and pleocytosis are evidence of inflammation, it does not follow *a priori* that they are dependent one

on the other or must be present together. They are quite different elements. The same is true of the Wassermann in relation to globulin and albumin.

COLLOIDAL GOLD TEST

The chemical nature of the substance which produces the colloidal gold reaction is not clearly defined. The colloidal gold reaction is a method of differentiating albumins and was used by Lange in the endeavor to classify the type of globulin occurring in the spinal fluid. Without having succeeded in making this classification, he did discover this practical test. An obvious assumption is that the test is dependent on the albumins or globulins in the spinal fluid. It is probable that the actual type of reaction obtained is dependent to some extent on the balance of globulin and albumin present in the fluid. However, it seems to be a fact that it is not the globulin ordinarily present that causes the precipitation of the gold. Matskiewitsch attempted to identify the substance causing the colloidal gold reaction as peptone. Weston disagrees with this finding. Our clinical experience has shown that the gold reaction may be obtained with a fluid that gives no precipitation with half saturated ammonium sulphate.

Our assumption in this consideration is that these substances may appear independently one of the other, and that while on the whole they represent reaction substances of inflammation, they are in some respects entirely different responses.

SPINAL FLUID FINDINGS

Now if this be so, that these substances may occur independently one of the other (with the one exception that globulin means an increase of albumin) it would follow that a number of combinations can occur, and as a matter of fact do occur. Our evidence is derived from two sources—the routine examination of several thousand fluids and examination of spinal fluids from cases of neurosyphilis undergoing treatment.

From the first group are obtained instances of the independent appearance of the several elements and their appearance in various combinations. From the cases under treatment one finds that one test or another disappears first, and finally only one may be left positive. The different combinations appearing in these two groups are shown in the accompanying table.

Brief examples may be given of the reactions occurring in Group 1. The Wassermann reaction occurs independently of other tests, not infrequently in the endarteritic forms of cerebral syphilis. It occasionally is found as the only positive test in cases of congenital

RESULTS OF SPINAL FLUID TESTS

Spinal Fluid Finding	Condition in Which This Finding Occurred (Untreated Cases)	Findings Occurring in Treated Cases of Neurosyphilis
Pleocytosis	Congenital syphilis, undiagnosed mental disease	
Albumin	Trauma, Korsakoff's psychosis, undiagnosed mental disease	
Wassermann reaction	Vascular neurosyphilis, congenital syphilis	General paresis
Colloidal gold test	Undiagnosed condition	General paresis, tabes, etc.
Pleocytosis and albumin		
Pleocytosis and Wassermann reaction.	.....	General paresis, cerebrospinal syphilis
Pleocytosis and colloidal gold test	.....	General paresis
Globulin and albumin	Cerebrospinal syphilis, trauma, cerebral hemorrhage, brain tumor, chronic alcoholism	General paresis
Albumin and Wassermann reaction		
Wassermann reaction and colloidal gold test	.....	General paresis
Albumin and colloidal gold test	Undiagnosed mental disease	Cerebrospinal syphilis
Pleocytosis, globulin and albumin		
Pleocytosis, albumin, Wassermann reaction	Cerebrospinal syphilis	General paresis
Pleocytosis, albumin, colloidal gold test		
Pleocytosis, Wassermann reaction, colloidal gold test	Neurosyphilis	
Globulin, albumin and Wassermann reaction	.....	General paresis, cerebrospinal syphilis
Globulin, albumin and colloidal gold test	Multiple sclerosis	Tabes, cerebrospinal syphilis
Albumin, Wassermann reaction and colloidal gold test		
Pleocytosis, globulin, albumin and Wassermann reaction.		
Pleocytosis, albumin, colloidal gold test and Wassermann reaction	.....	General paresis
Pleocytosis, globulin and colloidal gold test	Multiple sclerosis, brain tumor, apoplexy	General paresis
Globulin, albumin, Wassermann reaction and colloidal gold test	General paresis, cerebrospinal syphilis	General paresis
Pleocytosis, globulin, albumin, Wassermann reaction and colloidal gold test	General paresis, tabes, cerebrospinal syphilis	General paresis, tabes, etc.

syphilis, tabes and diffuse neurosyphilis. On the other hand, we have seen cases in which it was the only test to be absent. This of course is always true in nonsyphilitic inflammatory conditions giving a positive reaction for the other tests. We have also seen it even in cases of general paresis, tabes and congenital syphilis.

Pleocytosis is the most variable of the abnormal findings. It is not infrequently absent in the chronic inflammatory processes, e. g., in general paresis, tabes, brain tumor and multiple sclerosis, when the other tests are positive. On the other hand, it may be present in the absence of all other tests. For example, a woman suffering from glycosuria and a mild depression showed twelve cells per cubic millimeter; a second puncture showed sixteen cells per cubic millimeter. Her husband was a tabetic. A second example is that of a 20-year-old boy with all the signs of congenital syphilis, with outbreaks of violence, confusion and amnesia. He showed fifty-six cells per cubic millimeter, the other spinal fluid reactions being negative.

An increased amount of albumin as the only abnormal finding is not infrequent. In many instances where one finds this as the only evidence of pathology, one is at a loss to be able to interpret it, and we have seen frequent examples of this sort in dementia praecox and unclassified psychoses. According to Myerson an increased amount of albumin is common in Korsakoff's psychosis. I have been able to confirm this in a very small minority of Korsakoff cases. It also occurs after fractured skull and after fairly recent hemorrhage.

The gold reaction may occasionally be found as the only positive test, though this has been quite rare in our experience. We have found it in a few psychotic cases in which we were unable to explain its significance and in a few cases of syphilis.

It seems unnecessary to go into detail as to the combination of these findings with their clinical significance. It suffices to indicate on the table in what conditions we have found these to occur.

In cases of neurosyphilis receiving intensive antisyphilitic treatment, there usually is a change in the laboratory tests. We find that there is no general rule as to which test is the first to become weakened or to disappear, or as to which will remain positive. As one would expect, they do not all change at identically the same time. Thus in cases of general paresis undergoing treatment the Wassermann reaction is frequently unchanged despite long and intensive arsenic and mercury injections and may remain positive after all other tests have become normal. In another case with the same diagnosis and similar spinal fluid findings before treatment, the Wassermann reaction may be the first test to become negative. The same variation is to be found with the colloidal gold test, globulin and albumin.

In the table (third column) we have indicated the combination of findings that we have seen in treated neurosyphilitic cases. In addition a few examples are given in more detail.

REPORT OF CASES

OBSERVATION 1.—*Wassermann Reaction Remained Positive—All Other Tests Became Negative.*—Woman, 57 years of age, diagnosed as a case of general paresis.

Before treatment: Wassermann reaction +; globulin +++; albumin +++; cells 22 per c.mm.; colloidal gold test, general paresis.

After treatment: Wassermann reaction +; globulin 0; albumin negative; cells 0 per c.mm.; colloidal gold test, negative.

OBSERVATION 2.—*Colloidal Gold Test Remained Positive—All Other Tests Became Normal.*—Diagnosis: General paresis.

Before treatment: Wassermann reaction +; globulin +++; Albumin +++; cells 37 per c.mm.; colloidal gold test, general paresis.

After treatment: Wassermann reaction —; globulin 0; albumin negative; cells 3 per c.mm.; colloidal gold test, general paresis.

OBSERVATION 3.—*Pleocytosis and Wassermann Reaction Remained Positive; Other Tests Negative.*—The patient was a man of 35 with numerous neurasthenoid complaints, blood and spinal fluid positive to all tests. After two years of treatment spinal fluid findings were: Wassermann reaction +; globulin 0; albumin negative; cells 110 per c.mm.; colloidal gold test negative.

OBSERVATION 4.—*Globulin and Albumin Remained Positive; Other Tests Negative.*—Diagnosis: General paresis.

Before treatment: Wassermann reaction +; globulin +++; albumin +++; cells 90 per c.mm.; colloidal gold test 5 5 5 5 5 5 3 0 0 0.

After treatment: Wassermann reaction —; globulin ++; albumin ++; cells 3 per c.mm.; colloidal gold test negative.

OBSERVATION 5.—*Colloidal Gold Test and Wassermann Reaction Remained Positive; Other Tests Became Negative.*—Diagnosis: General paresis.

Before treatment: Wassermann reaction +; globulin +++; albumin +++; cells 22 per c.mm.; colloidal gold test, general paresis.

After treatment: Wassermann reaction +; globulin 0; albumin negative; cells 0 per c.mm.; colloidal gold test, general paresis.

OBSERVATION 6.—*Albumin and Colloidal Gold Test Remained Positive; Other Tests Became Negative.*—Diagnosis: Cerebrospinal syphilis.

Before treatment: Wassermann reaction +; globulin +++; albumin +++; cells 11 per c.mm.; colloidal gold test 5 4 3 2 2 1 0 0 0 0.

After treatment: Wassermann reaction negative; globulin 0; albumin +; cells 3 per c.mm.; colloidal gold test 0 1 3 4 3 1 0 0 0 0.

OBSERVATION 7.—*Globulin, Albumin and Wassermann Reaction Remained Positive—Pleocytosis and Colloidal Gold Test Became Negative.*—Diagnosis: General paresis.

Before treatment: Wassermann reaction +; globulin +++; albumin +++; cells 45 per c.mm.; colloidal gold test 5 5 5 5 5 5 5 2 0 0.

After treatment: Wassermann reaction +; globulin ++; albumin ++; cells 4 per c.mm.; colloidal gold test negative.

OBSERVATION 8.—*Globulin, Albumin and Colloidal Gold Test Remained Positive—Wassermann Reaction and Pleocytosis Became Negative.*—Diagnosis: Cerebrospinal syphilis.

Before treatment: Wassermann reaction +; globulin + + +; albumin + + +; cells 56 per c.mm.; colloidal gold test, 0 0 1 3 3 2 1 0 0 0.

After treatment: Wassermann reaction negative, globulin +; albumin +; cells 0 per c.mm.; colloidal gold test 2 2 2 3 3 1 0 0 0 0.

OBSERVATION 9.—*Wassermann Reaction Became Negative; Other Tests Remained Positive.*—Diagnosis: Cerebrospinal syphilis.

Before treatment: Wassermann reaction +; globulin + + +; albumin + + + +; cells 41 per c.mm.; colloidal gold test, general paresis.

After treatment: Wassermann reaction —, globulin + +; albumin + +; cells 9 per c.mm.; colloidal gold test, syphilitic.

OBSERVATION 10.—*Pleocytosis Reduced to Normal; Other Tests Remained Negative.*—This is a very frequent finding.

Even though not a part of this discussion, we would like to add that the Wassermann reaction in the blood serum varies independently of the spinal fluid findings. Thus it may become negative while all the spinal fluid tests remain unchanged or it may remain positive when all the spinal fluid tests become negative. It is also pertinent to note that the changes in these spinal fluid tests do not always parallel clinical changes in the patients. Thus we have seen illustrations where although the tests became negative the symptoms were in no way improved; and on the other hand, where there was marked symptomatic improvement without any change in the tests.

SUMMARY

Of the five spinal fluid laboratory tests commonly used, only cells and albumin are normal constituents of the fluid, and the excess amounts of these are evidence of pathology. The Wassermann reaction is for practical purposes specific for syphilis of the nervous system. Pleocytosis, globulin, albumin and colloidal gold reaction are generally indicative of an inflammatory reaction of brain, cord or meninges. These tests may be positive owing to infection, mechanical injury, tumor, trauma, vascular insults, multiple sclerosis, etc. As a rule, globulin, albumin increase, pleocytosis and a positive colloidal gold reaction occur together and when the Wassermann reaction is positive, it is usual for the other four to be positive also. Although usually present together and in a general way indicative of the same pathologic condition, each reaction is produced by a distinct chemical element which may be present alone (except that theoretically globulin means an increase in the total amount of albumin, as globulin is a special albumin). Thus one may find the Wasserman reaction positive, all other tests negative, a positive colloidal gold reaction as the only positive finding, only a pleocytosis or merely an albumin increase.

Further, they may occur in various combinations. This is theoretically possible on the basis of difference in chemical constitution, and it is shown in this paper that this actually does occur.

Additional evidence of the independence of each element is offered in the result of treatment of cases of neurosyphilis. Starting with all tests positive, it is shown that they become negative often one at a time and the different combinations are left positive, or only one is left positive.

CONCLUSIONS

1. There is a nonconcomitancy of the inflammatory elements of the spinal fluid commonly tested for in diagnosis of disease of the central nervous system.

2. Any one may be present or absent when the others are present—with the exception that globulin presages an increased amount of albumin.

3. No spinal fluid can be considered negative in which all these tests have not been used.

4. No one element tested for contains the element or fraction that gives another test, except that the total albumin contains the globulin fraction, in part at least.

5. In neurosyphlitic cases receiving treatment these substances disappear at differing rates which vary in different cases, so that no general law can be laid down as to which element is most easily affected by treatment in any particular case, though in general the pleocytosis disappears first.

6. The presence or absence of these products of inflammatory reaction does not always parallel the clinical change in the treated neurosyphilitic patient.

INFLUENZA PSYCHOSES IN SUCCESSIVE EPIDEMICS *

KARL A. MENNINGER, M.S., M.D.
TOPEKA, KAN.

Psychoses following influenza, as precipitated by the epidemic of 1890-1892 and reprecipitated by the epidemic of 1918-1919, make a particularly interesting study. Among the nearly 200 cases of post-influenzal psychoses studied at the Boston Psychopathic Hospital during the past year, only two such cases were observed. A third case, in which a psychosis had followed the earlier epidemic, was received at the hospital during this period, but without the history of recent influenza. These three cases are reported below.

These cases are interesting because of the question of specificity which Kraepelin[1] raised. He maintained that each acute infectious disease was probably capable of producing mental symptoms of a peculiar sort, a virtual "influenza psychosis," "typhoid psychosis," etc. This view has never been well supported, and as yet we know only that the psychoses following influenza are numerous and of great variety.[2] This might connote a quantitative specificity as contrasted with Kraepelin's qualitative specificity. In the case of many, some writers[3] say nearly all persons, influenza has been followed by states of depression, irritability, irascibility, deconsolateness, etc., which undoubtedly represent *formes frustes* or mild forms of the psychoses appearing in bolder form in less fortunate subjects.

Why some persons should be particularly subject to the neurotoxin of influenza as these cases here would indicate, is as yet unexplained. In the cases cited below there must have been, during the three decades intervening between the two attacks of influenza, both of which were followed by such striking mental reactions, many incidents and agents capable of more or less psychopathologic damage; yet

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* This is the fourth of a series of papers on influenza and mental diseases from the Boston State Hospital, Psychopathic Department, Series 1919.

1. Kraepelin, E.: Kraepelin advanced this theory during his early work on the "symptomatic psychoses," but summarizes his views in his Psychiatrie, ed. 8, Leipzig and Vienna, 1912.

2. Menninger, Karl A.: Psychoses Associated with Influenza, II. Specific Data. An Expository Analysis, Arch. Neurol. & Psychiat. **2**:291 (September) 1919.

3. Pritchard: Nervous Diseases and Psychoses Following the Grippe, Internat. Clin. XI, ser. 5; Osler: Practice of Medicine, New York.

apparently no cause produced a result such as was wrought for the second time by influenza, in the short course of a few days.

Another significance of these cases lies in their bearing on the relationship of the infection (toxic) psychoses to schizophrenia (dementia praecox). Postfebrile delirium and schizophrenia were by far the most numerous psychiatric sequelae of influenza.[4] There is, moreover, the notorious[5] similarity in symptomatology of catatonia and hebephrenia to the "postfebrile deliria." On this, in part, is based the thesis, by no means new, that delirium and some forms of schizophrenia may have a similar pathologic basis, and hence etiology. Cases such as those I present, in which an infection (influenza) or some other incident is followed by a psychosis which improves, only to reappear under renewed conditions of deleterious influence (here influenza, except in the third case) suggest the chemical process of reversibility, and specifically the reversibility or partial reversibility of schizophrenia. By extension of the hypothesis of the similar etiology of delirium and schizophrenia, and comparison with the phenomenon just noted, one can conceive that postfebrile deliria may represent, as the writer would propose, a form of reversible schizophrenia.

REPORT OF CASES

CASE 1.—A man, aged 49, with an unfavorable family history, was normal until he had an attack of influenza at 25. He was psychotic for a short time afterward, and had been queer for the past few years. He has had frank psychosis with conduct disorder since he had influenza in 1918.

Family History.—This embraces a psychotic father ("asocial, queer, brooded, suicidal"), an insane cousin, other "high strung" cousins, and a mother who had "a nervous breakdown." He was an only child.

Personal History.—He was not considered queer, asocial, stupid or abnormal either before or after the first attack of influenza. The medical history was negative; his habits were good. He did not marry.

First Attack of Influenza.—"About twenty-five years ago" he is known to have had influenza at the time of "an epidemic." Thereafter he was for a time "affected in his head," at least for a few months. In subsequent years he was very irregular at work, changing jobs often, usually without adequate cause.

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4. Fell, E. W.: Postinfluenzal Psychoses, J. A. M. A., **72**:1658 (June 7) 1919; Burr, C. W.: Mental Complications of Influenza, Med. Clin. of N. America **2**:709 (November) 1918; Harris, A. F.: Influenza as a Factor in Precipitating Latent Psychoses, Boston M. & S. J. **158**:22 (May 29) 1919; Menninger, Karl A.: Psychoses Associated with Influenza, I. General Data: Statistical Analysis, J. A. M. A., **72**:235 (Jan. 25) 1919.

5. A point made by many writers, old and new. For example, see the textbooks of Kraepelin, Binswanger, Siemmerling, Paton, Regis; the monographs of Bonhoeffer and of Bleuler; numerous abstract discussions such as those of Hoch, Deny, Dide and others, and finally, the recent clinical observations and reports of Austregesilo, Harris, Fell, the writer and others.

For some years he has been rather paranoid, suspicious and queer, and has begun to do peculiar things, which, however, have been overlooked. Fits of anger and destructiveness, with attitudinization have also been noted. These symptoms have developed gradually during the last seven years, but they did not become serious until after the influenza.

Second Attack of Influenza.—This occurred in October, 1918. Thereafter his psychopathic condition became much worse. He manifested (for the first time) delusions and hallucinations, enfeebled intellectual processes and an emotional tone varying from silly euphoria to causeless agitation. Silliness was manifested in manner, speech and conduct.

Physically, he showed unequal pupils, slurred speech, hypo-active left ankle jerk, and right facial paresis, but otherwise nothing abnormal. All laboratory findings, including serum Wassermann and spinal fluid, were negative.

At first the case was considered, *a priori,* one of neurosyphilis, but laboratory findings disproved this, and the patient was considered merely hypophrenic. (His psychometric rating was 8.7 years, variation total of 17). However, increasing symptoms changed the diagnosis. He became more and more silly; he sat about the ward in fixed positions and without speaking a word for days at a time; he became mute and totally inacessible. He voiced delusions of being "butchered" and "put in straight jackets." He became steadily worse mentally and was transferred, after six weeks' observation, to the Boston State Hospital.

Diagnosis.—Schizophrenia hebephrenica.

Case 2.—A woman, aged 37, had influenza at 10 followed by psychosis and commitment to an asylum for ten years. She absconded and lived at large, being self-supporting for ten years, when a second attack of influenza (1918) precipitated symptoms requiring commitment.

Family History.—She had an alcoholic father and a sister, now 45, who has recently been committed to an asylum as insane; she is self-accusatory and depressed.

Personal History.—Birth and childhood were negative. She progressed to the third grade, and was considered healthy. She had diphtheria.

First Attack of Influenza.—At the age of 10 (1891) she had influenza. Not many details are known, save that thereafter she was "different"—she was "strange, contrary, ill natured and did not get on well with other children." She claimed that she was discriminated against, that she was abused, that her employers persecuted her and that people were jealous of her.

Finally, she was committed at about 17 years of age. She remained in the state hospitals for some ten years; then she absconded, and was taken in by some family. A friend of her family got in touch with her and heard from her frequently. She lived at large with some fair degree of success, but changed employment very frequently. This continued for ten years.

Second Attack of Influenza.—She was taken to the City Hospital with influenza, Jan. 18, 1919. She recovered and was up and about the ward, but "made irresponsible statements and was excitable." She was transferred to the Boston State Hospital.

She expressed great perturbation over being here, and hastened to assure every one that she was quite free from any mental trouble. She cooperated poorly and was evasive, arrogant, egotistic and irritable. She was seclusive, irascible and unpopular with the patients, and was antagonistic toward everything—physicians, nurses, baths, occupational therapy, etc. She was notably dis-

interested, and showed a poverty of ideation and emotion. No delusions or hallucinations were elicited.

Her psychometric rating was 10.3, with a variation total of 15, which is, of course, quite high. She kept saying, "Now I want you to tell me if it is not correct, for I'm perfectly willing to verify it."

Physical examination and laboratory findings, including fluid findings, were entirely negative.

Diagnosis.—Schizophrenia hebephrenica.

CASE 3.—A woman, aged 48, supposedly well educated until she had an attack of influenza in 1890, thereafter was gravely demented, but was useful as a domestic. In recent years she has become increasingly queer, and finally totally incompatible. This is a typical case of old schizophrenia. She probably did not have influenza in the recent epidemic.

Family History.—Nothing abnormal was revealed.

Personal History.—She is said to have been "a very well educated woman, and perfectly normal until she had the influenza during the epidemic of 1890."

First Attack of Influenza.—"The influenza struck her brain." She was "just like a child . . . quiet . . . never speaks unless spoken to. Wasted her money and threw away or tore up what she bought. Always talked incoherently." She was employed for the past twenty years by a family from whom this (the only) information was obtained.

Present Episode.—There is no record of her having had influenza during the 1919 epidemic. For a year she had been acting more and more strangely, ceased bathing herself, began to answer her mistress irrelevantly, etc. On the day of admission she "suddenly began to kick the puppy" and frightened her mistress, so that she was brought to the Psychopathic Hospital.

Mental examination showed marked dilapidation of all thought processes; disorientation, delusions of transformation, hallucinations, profound apathy, hypokinesis, and occasional resistiveness and negativism.

Physical examination and laboratory findings were wholly negative.

Diagnosis.—Schizophrenia hebephrenica.

A BRIEF RÉSUMÉ OF NEUROLOGIC AND PSYCHIATRIC OBSERVATIONS IN A HOSPITAL CENTER IN FRANCE

JUNIUS W. STEPHENSON, M.D.
Chief of Clinic, Second Division, Neurological Institute; Adjunct Visiting Neurologist, Bellevue Hospital; Visiting Neurologist, Cornell Dispensary
NEW YORK

This résumé includes a statistical report, with brief citations and conclusions drawn therefrom, of cases observed at Hospital Center Mar-sur-Allier, France, during the period from September 18 to Dec. 31, 1918. During that period approximately 25,000 patients were admitted to the Center. They were received as early as forty-eight hours after engagement. At the time of the armistice there were operating in the Center seven hospitals with individual capacity of 2,000 patients, and a convalescent camp with a capacity of 5,000 patients. Each hospital and the convalescent camp had its individual psychiatrist. Each hospital designated a special ward for functional and psychiatric cases. Brain, spinal cord and peripheral nerve injuries were not designated a special ward, but were seen and diagnosed by the respective neurologists.

The total number of neurologic and psychiatric cases observed was 507, classified as follows:

TABLE 1.—NEUROLOGIC AND PSYCHIATRIC CASES

Brain injury	20
Spinal cord injury	17
Cerebral concussions	54
Spinal cord concussions	8
Peripheral nerve injuries	266
Neuroses	103
Psychoses	23
Epilepsy	6
Mental defectives	10

BRAIN INJURIES

Of eighty patients admitted to Base Hospital No. 48 with gunshot wounds of the skull, ten, or 12.5 per cent., showed evidence of injury to the brain substance in these locations:

TABLE 2.—CASES OF BRAIN INJURIES

Frontal	3
Parietal (motor, sensory, aphasic symptoms)	3
Parietal (motor symptoms only)	1
Occipital	3

All of the occipital injuries resulted in infection, two developing abscess of the occipital lobe, and one a purulent meningitis at the site of a previous decompression. (One occipital case in Base Hospital No. 14 also developed an abscess). One frontal injury resulted in an abscess. The great frequency of occipital infection was a matter of interest, perhaps explained by the great vascularity of this area.

I was particularly impressed by two objective symptoms which were present in the frontal cases, symptoms which I had previously observed in civil practice. These symptoms were "stuporous states" and "yawning." They were not observed at all in the parietal cases nor was there any yawning in the occipital cases, and stupor appeared only just before death.

REPORT OF CASES

CASE 1.—*History.*—One of the occipital cases was in reality one of late abscess and was quite interesting. This soldier was admitted as an ambulatory patient. There was a closed wound over the left occiput and from a pin-point opening in the lower angle of the wound exuded a very slight amount of serous fluid. He was most carefully examined for evidence of hemianopsia, but none was found. For several days he roamed around like any other convalescent wounded patient, with no complaints whatsoever.

Course and Treatment.—Suddenly one day he complained of intense headache. His temperature at 2 p. m. was reported to be 104 F. I saw him about 9 p. m. the same evening, and his temperature was 99.5 F. His headache had disappeared, and he said he felt "all right." On examination he showed complete left homonymous hemianopsia. This was the only objective finding. The next morning his headache had entirely disappeared and he felt quite well. The hemianopsia had entirely disappeared. For ten days thereafter there were absolutely no objective or subjective symptoms. He was repeatedly examined for hemianopsia and none was found. His headaches then suddenly reappeared as did also his hemianopsia. Vomiting became a distressing symptom; his condition progressed from bad to worse. Surgical exploration revealed an abscess of the left occipital lobe. He died seven days later and necropsy revealed the abscess and beginning purulent meningitis. The interesting feature in this case was the behavior of the hemianopsia, which can probably be explained on a vascular basis.

CASE 2.—*History.*—A brief notation in regard to the frontal abscess case. This soldier was admitted in a stuporous state, but during the first few hours could be aroused to take nourishment. He "yawned" considerably. All the deep reflexes were normal, the abdominals and plantars were also normal. He died seven hours after admission.

Necropsy.—This revealed fracture of the left frontal bone, the entire left frontal lobe a pulpy abscess mass; petechial hemorrhages; no evidence of infection in the brain stem; a large "spread out" blood clot over the right occipital lobe; slight reddening of the anterior border of the left parietal lobe, but no petechial hemorrhages or evidences of infection within it. There was no communication established between these various lesions. It will be observed that the direction of the force of the trauma was from before downward, inward and backward. Apparently there were three distinct pathologic conditions: First, a fracture of the skull with direct injury to brain tissue

followed by infection and abscess; second, general concussion (commotion) causing the petechial hemorrhages; third, the contrecoup phenomenon manifested by the findings in the occipital region.

SPINAL CORD INJURIES

The location of these injuries was about equally distributed between the dorsal, lumbar and sacral regions. I saw no cervical injuries. Where there was complete section of the spinal cord death intervened usually from local infection, or infection involving the genito-urinary tract. Death occurred earlier in mid-dorsal regions than in any other location.

CEREBRAL CONCUSSIONS

On this subject some probably will take issue with me. However, the cases here classified as concussion presented identical symptomatology, namely, occipital pain, mental confusion of varying degrees, mental and physical fatigability, following a definite history of a period of unconsciousness varying from one to twenty-four hours, resulting from explosions. No external injuries were present. Owing to the late period at which these cases were received, lumbar puncture was of little assistance. In a majority of the cases there was an overfilling of the retinal vessels and in some there was clouding of the nasal half of the optic disk and a strong suggestion of weakness of both sixth cranial nerves. Except for hyperactivity of the deep reflexes and fine rhythmic tremors, further neurologic examination was negative. In this class there was no attempt at "conversion," nor could any conscious or subconscious desire or fear be elicited. It was significant that after the cessation of hostilities the number of patients of this class arriving in this Center was considerably diminished.

The value of lumbar puncture at the earliest possible moment in suspected cases cannot be overestimated. In acute confusional cases of any degree, early performed, it will often eliminate a purely functional or psychic condition, and, inasmuch as concussion cases often develop the syndrome of traumatic neurasthenia, the early and routine use of lumbar puncture as a diagnostic aid will often protect these sufferers from being stigmatized "fakers" or seekers after money.

SPINAL CORD CONCUSSION

As to the existence of clinical phenomena following concussion of the spinal cord all are agreed, but as to the actual pathologic tissue condition, there is a difference of opinion. I am inclined to support the theory of necrosis as promulgated by H. Claude and J. Lhermitte. Corroborative evidence of this assumption is furnished by the fact that although lumbar puncture was performed promptly in the majority of our cases, the result of examination of the fluid was negative. The

one necropsy which we were able to get revealed no macroscopic abnormalities in the cord. The pronounced clinical disturbance made a hemorrhage, later absorbed, most unlikely.

REPORT OF CASES

CASE 1.—Capt. X, thrown several feet by the explosion of a shell, received no external injury. On admission to hospital he suffered from incontinence of urine and feces and a spastic paraplegia with complete anesthesia to the level of the tenth dorsal vertebra. Roentgen ray and lumbar puncture were negative. The incontinence disappeared within twenty-four hours, followed by gradual disappearance of sensory and spastic phenomena.

CASE 2.—B., who had received a gunshot wound in the lumbosacral region and was admitted to hospital as an ambulatory patient, presented typical drop-foot gait, and gave a definite history of rectal and vesical incontinence. Examination revealed a double drop-foot, and knee jerks were present and equal. The ankle jerks were not elicited and there was hypothesia over the entire sacral segment on both sides. Lumbar puncture was negative. Roentgen ray revealed fracture of the transverse process of the fourth lumbar vertebra and "tips of lower spines." He improved steadily, and was later discharged for limited service.

TABLE 3.—PERIPHERAL NERVE INJURIES

Nerves Injured	No. Cases	Percentage, Approximately
Median	41	15
Ulnar	45	16
Musculospiral	47	17.5
Median and posterior interosseous	1	
Median and musculospiral	6	
Internal cutaneous	2	
Musculocutaneous (upper)	4	
Circumflex	3	
Brachial plexus	20	
Sciatic trunk	33	12.5
External popliteal	39	14.5
Internal popliteal	1	
Internal saphenous	4	
Anterior crural	6	
Small sciatic	1	
Anterial tibial	4	
Median and ulna	7	
Sciatic trunk and internal saphenous	1	
Idiohypogastric and ilio-inguinal	1	

CASE 3.—Pte. C., a stretcher patient with a gunshot wound of the lumbosacral region, had a flaccid paralysis of both lower extremities with definite history of incontinence. (The first day in hospital it was necessary to catheterize the patient.) Examination revealed the left knee jerk absent, right knee jerk sluggish, both ankle jerks absent, no response to plantar stimulation, complete anesthesia to all sensation on the right side over areas corresponding to the distribution of the lower lumbar and entire sacral segments. On the left side there was a similar distribution of hypothesia. Within forty-eight hours after admission he began to improve, the first objective improvement being return of the right knee jerk. Improvement, motor and sensory, progressed rapidly but seven days after admission he developeed

double streptococcic pneumonia and died within twenty-four hours. On account of location and nature of the wound lumbar puncture was not performed, but necropsy revealed, in addition to the pulmonary findings, a machine gun bullet lodged against the fourth lumbar spine. The spinal cord and its membranes in situ and on section showed no macroscopic abnormality. There was no evidence of extravasation of blood within the spinal canal.

The total number of these injuries was 266.

The peripheral nerve injuries include all grades from slight irritative to complete. Often on admission a given nerve would have every evidence of a complete lesion, but in the course of a week or ten days regenerative phenomena would appear and proceed to partial or complete recovery. The time of appearance of regenerative phenomena was so constant—from one to two weeks after injury—that it is my opinion that one month is sufficient time for watchful waiting and that surgical intervention is then indicated. It was a common occurrence in thigh injuries to have the pitcure of sciatic trunk injury prove, in a week or ten days, to be isolated external popliteal injury. Causalgia was most distressing in the median injuries, to a less degree in the external popliteal. Ulnar causalgia promptly responded to treatment. The most common description of the pain was "It is so hot, burns like fire." It was quite common to see a soldier with hand enveloped in a cloth which he frequently moistened with cold water.

In considering the prognosis of peripheral nerve injuries, muscle tone proved so vastly superior to other signs that I practically relied on it, abandoning electricity. In this connection I might also state that I abandoned the use of electricity in the treatment of irritated, concussed and compressed nerves, relying on massage and passive movements. The occurrence and rapidity of regeneration of nerves was in the following order: Ulnar, musculospiral, anterior tibula, external popliteal, median, sciatic trunk. Peripheral nerve injuries occurred in approximately 6 per cent. of the extremity wounds.

NEUROSES

Total number of these cases was 103.

TABLE 4.—CASES OF NEUROSES

Hysteria	37
Neurasthenia	15
Psychasthenia	5
Unclassified psychoneuroses and constitutional psychopathic states	46

The hysterias include the monoplegias, paraplegias, "gross shakers," "nodders," mutisms and other forms of conversion. The neurasthenias include those neurasthenic states commonly seen in civil life, a large proportion of which gave a fairly definite history of having been con-

cussed. I saw but one case of real neurasthenia. The psychasthenias all showed actual obsession. The other psychoneuroses and constitutional psychopathic states were the emotional, unstable, apprehensive, egotistical personalities. The term "gross shaker" is used to designate those cases showing gross rhythmical tremors of the entire body, not unlike that observed in astasia abasia, whereas the term "nodder" applies to those with atypical torticollis-like movement of the head and neck. Fine rhythmic tremors were demonstrable in all the psychoneurotic as well as in the neurasthenic patients. The usual exaggeration of deep reflexes was present. I saw no case of complete hemianesthesia, the affected extremity alone being anesthetic. Contraction of the visual field was rare. Of the total number of functional cases, two only had received actual wounds. One patient, a mental defective, was a "gross shaker" whose wound necessitated amputation at the thigh. His "shakes" disappeared long before the wound healed. The other patient developed a hysterical monoplegia after his wound had healed.

Besides the elements of fear and desire in the development of the neuroses, I am convinced that there were two factors which played a potent part in our cases; namely, physical exhaustion and the suggestion offered by the notoriety given so-called "shell shock" by the press of the United States. One of the phenomena of fatigue is tremor, and it was in the "shakers" or tremor cases that exhaustion was especially operative, the tremor developing as the result of exhaustion. Then, the soldier being too slowly evacuated, sufficient time elapsed for the elements of fear and desire to come into action, producing exaggeration and more or less chronicity. The "nodding" may have been, and probably was, a defensive reaction. Nevertheless, the subsequent events are at least strong, presumptive evidence that the element of exhaustion was the fundamental starting point of the existing condition.

The baneful influence of press notoriety was demonstrated in the psychoneurotic and constitutional psychopath. A large proportion of these patients always knew the nature of their trouble. It was "shell shock," and if perchance someone had unthinkingly written the words "shell shock" on the diagnosis tag or field card, it was doubly "shell shock." Of course, in these cases the element of fear or desire could be elicited, but my contention is that the suggestion offered by the stories of "shell shock" was a most fertile field for the development of this fear or desire. These were the emotional, anxiety states. The soldier would frankly say that he was all wrought up; he wanted to go back, but didn't know whether or not he could stand it; he was afraid he would go to pieces, etc., and in his behalf I must say I had

every reason to believe he was sincere. He had read of the ravages of "shell shock," which enhanced his fear.

A few cases of anxiety states seemed to be of endocrine origin as shown by classical tremors, von Graef, tachycardia and the various skin reactions with, in some instances, more or less enlargement of the thyroid gland. These cases rapidly improved on rest in bed, but on any stress or exertion the symptoms would return.

In fully 90 per cent. of the functional cases there existed an underlying predisposition. It was in the exhaustion cases that this predisposition could not be elicited. The large majority of patients developing hysterical palsies gave a definite history of previous episodes. The usual story of the aphonic or mute, was that he had been gassed. Here the element of exhaustion apparently was insignificant while that of fear or desire could usually be elicited.

PSYCHOSES

These cases presented nothing out of the ordinary. Dementia praecox predominated. It will be observed that no case of delirium tremens was seen. Nor was there any case of abstinence symptoms as the result of drug addiction.

TABLE 5.—Cases of Psychoses

Dementia praecox	13
Manic depressive	6
Exhaustion	4

MENTAL DEFECTIVES

The number of mental defectives is given as ten. Undoubtedly there were more. This number does not include the total number examined for possible defective mentality, the fact being that equally as many, if not more, were found to be in reality not mental defectives, but uneducated boys, and this was particularly true of those coming from a certain section of the United States.

No résumé would be complete without acknowledging the hearty cooperation extended the neuropsychiatric department of the Center by the commanding officer of the Center, Col. George S. Skinner, M. C., and the assistance given by the individual unit commanders. I wish to express my warmest appreciation of the untiring cooperation and assistance rendered by the neuropsychiatrist of the individual hospital units whose efforts made for neurology and psychiatry a most creditable showing in the Hospital Center Mar-sur-Allier.

20 West Fiftieth Street.

Abstracts from Current Literature

A CONTRIBUTION TO THE EXPERIMENTAL STUDY OF THE LESIONS OF SHOCK. A. MAIRET and G. DURANTE, Rev. neurol. **26**:97, 1919.

During the late war, the attention of neurologists was focused on accidents resulting from the detonation of powerful explosives, at short distances, without producing signs of external injury. The polymorphism of the lesions and the great variety of symptoms encountered in these conditions, as well as their pathogenesis, has given rise to many conflicting opinions.

Some observers consider the syndrome of shock to be purely functional and entirely different from the syndrome of emotion. Others believe both of these syndromes to be due to organic lesions, the exact nature of which is still to be determined. The lack of sufficient pathologic material has been the cause of contradictory opinions, and has led Mairet and Durante to expose a number of rabbits to powerful explosions at short distances, under conditions as nearly as possible like those met with in actual trench warfare, and to study the effects of such explosions.

In the first series of experiments (February, 1917) they placed rabbits in a cage on the ground, at the same level with the explosive, at distances varying from 1 to 2 meters. The explosives consisted of 135 gm. of melinite, which was later increased to 1,800 gm. of chedite and melinite.

Explosions with the lighter charges, in the open air, had no effect on the animals, but those with the heavier charges, and coming from a ditch, were much more efficacious. One of the animals became very weak, it fell down and lay in the cage, holding its head at first on the right and later on the left side. Examination revealed anesthesia of the cornea and extremities; it died thirty minutes later, but showed no gross lesions of the nervous system. The surviving animals seemed to suffer from dyspnea, which was temporary only and appeared to be due probably to the inhalation of toxic fumes from the explosives.

In another series of experiments (March, 1917) the arrangement was such that the explosions always occurred on the ground, the animals being at a distance of 2.5 meters from the charge, and suspended so that they received the shocks of the explosions at angles varying from 20, 30, 40 and 50 degrees.

In the last series of experiments (June, 1917) the authors utilized cross-trenches modeled exactly like those at the front. The explosives were placed on an iron bar attached to the upper opening of the trench. The animals were on top of the parapet (like a man in the reclining posture) or on a bench (like a man sitting); their position was such that they would receive the effects of the explosion directly, and at the same time would not be injured, in case they fell down.

Of eighteen animals experimented on, three were hit by fragments of explosives and were excluded from further study. Among the remaining fifteen, one was found to be dyspneic; some of them appeared shocked and excited, others feeble and depressed. Nearly all of them were breathing rapidly, for almost half an hour after the explosion. Most of them showed a temporary

myosis. The final outcome was out of proportion to the apparent gravity of the early symptoms of shock. Some of the animals remained on their backs motionless, with their paws in extension, but they gradually recovered from these symptoms and at the end of twenty-four hours seemed to be as alert and as contented as they had been prior to the experiment. One rabbit, which seemed to be affected very little immediately after the explosion, died at the end of twenty-four hours. None of the remaining animals showed any anesthesia, atrophies or paresis.

Early Lesions: Macroscopically, there was little difference in the lesions of the animals that died spontaneously and those that were killed a few days later. In all cases except one, the lungs were streaked with confluent foci of congestion involving one half or two thirds of each lobe. With the exception of one case of punctiform lacerations of the anterior border of the liver, due to a direct blow with a piece of explosive, the other viscera appeared normal.

The spinal cord presented slight hemorrhagic areas irregularly distributed on its surface, with a few small blood clots adherent to the roots. The brain appeared normal in some cases, and in others there were found small meningeal hemorrhages scattered on the upper surface of the cortex or in the region of the pons.

In one case there was a large cervical subcutaneous and submuscular ecchymosis.

The histologic lesions were quite uniform. In the lungs there were foci in which the alveoli were filled with blood, and the capillaries at the margins of these foci were markedly dilated. In two cases the kidneys showed hemorrhages along the external sheaths of the blood vessels; these were apparently due to the rupture of the small arterioles in the arched arteries. The uriniferous tubules and the glomeruli were normal. The liver, except in the case previously mentioned, was free from ecchymoses.

The spinal cord presented at one point or another slight hemorrhages in the pia. Hemorrhages in the roots, however, were common and more marked; they involved the interstitial tissue between the nerve fascicles, the latter remaining practically normal. Both the anterior and posterior roots shared in this involvement, which was always confined to their free or floating portions. The ganglions were normal. In the cord itself the vessels between the white fascicles were frequently surrounded by rather extensive hemorrhagic sheaths; these were rarely seen in the gray commissure or in the anterior horns. The motor and sensory cells were normal. The peripheral portion of the white fascicles did not always stain well with the Pal method. The nerve fibers appeared irregular and distended; the myelin was disintegrated and had lost its continuity of form. The axis cylinders were occasionally absent, always irregular, curled up and moniliform (beaded); they were frequently cut up, sometimes only punctiform in size, but occasionally extremely large. The lesions extend from the periphery to the center, and gradually disappear without any sharp line of demarcation. Acute primary degeneration, as described by Claude and Lhermitte, was observed in only two animals.

The lesions in the brain were somewhat similar to those in the cord, except that the superficial hemorrhages were less marked. These were confined mostly to the middle zone of the gray matter, where normally the vessels are surrounded by lymphatic sheaths, sufficiently large to separate the neighboring nervous elements; these sheaths, having been distended with blood after a vascular rupture, keep the blood confined within them, thus preventing com-

pression of the surrounding tissues allowing them to remain normal. Similar lesions were found in the pons and medulla. The central gray nuclei rarely showed any lesion whatever. Contrary to Mott's observation, the cells in the cortex and in the gray nuclei were perfectly normal. Finally, these authors observed circumscribed bruises on the surface of the cortex, with minute osseous fragments, which seemed to have been detached from the internal table of the bones of the skull. Were it not that they encountered the same lesions in the animals in which they studied the secondary effects of the explosions—"the late lesions"—they would have been inclined to attribute these to accidents at the necropsy.

Late Lesions: The animals intended for the study of these lesions were kept five, eight and nine months after they had been shocked. They were examined at frequent intervals and were found to be perfectly well, and without atrophy, paralyses or anesthesia.

Macroscopically they showed no evidences of any lesions in the cranium or nervous system. Histologic examination of the cord showed neither focal lesions, islands of necrosis nor cicatrices. The anterior horns, however, were irregular at the different levels; some were shorter and narrower than others. The number of motor cells was diminished and many of them were small without branched processes (dendrites). The lesions were diffuse and not destructive in nature, thus explaining the absence of paralysis and amyotrophy. There was no evidence of secondary degeneration of the white fascicles.

The Brain: In two of the animals there were seen numerous rounded tuberosities extending from the surface of the cortex to the angle of Bechterew's layer. These were lined with an incomplete layer of fibrous tissue and consisted of poorly stained, clotted material without nuclei; within this substance numerous vessels with thickened walls could be seen. The whole appearance of this lesion was that of a small cortical necrosis, into which the meningeal vessels, later, so to speak, included themselves.

Bechterew's layer and the layer of motor cells were riddled with vacuoles of various dimensions, giving the tissue an edematous appearance. The cells themselves were atrophied. These areas of vacuolar edema were not sharply outlined.

The motor cells were arranged in radiating columns, between which there were spaces free from cells. This arrangement of columns is due to a destruction of a certain number of radiating capillaries, which destroyed the cells depending on them for their blood supply. In some areas the changes in the motor cells were more marked; the cells were not distinct, they were arranged without order, and were often atrophied with small groups of well preserved cells interspersed between them. Localized meningeal adhesions, making the diffuse vascular changes more prominent, were also in evidence.

In a number of cases there were many areas filled with tracks of sclerosed neuroglia; these tracks were in the form of irregular bands or triangular spindles, which began at the cortex and extended to the zone of motor cells but did not reach the gray matter.

In two animals there were found, in a good many areas, perpendicular linear cicatrices breaking up the "geological" strata of the cortex. The authors think that the cicatrization is due to the passage of microscopic fragments of bone from the internal table of the skull. These foreign bodies are surrounded by tissue consisting of fusiform cells, arranged in the form of curly bands, and may occasionally be seen in the deeper subjacent white matter.

After reviewing in great detail the various mechanisms which have been called on to explain the pathogenesis of the lesions of shock, the authors express their belief that whereas the gross focal lesions give evidence that they are due to an actual contusion by the volume of gas resulting from an explosion, giving rise to bruising of tissue, the diffuse lesions of true shock are due to the effect of severe vibration, i. e., a general shaking up of the body; the selection of the organs involved depends on their anatomic and physiologic ability to withstand the phenomena of vibration.

In their conclusions, the authors insist on the necessity of reserving the term "shock" to the pathologic states which result from vibratory concussion or shaking up following the detonation of powerful explosives, the lesions of which are elementary, diffuse and without destruction or bruising of tissue.

The term "contusion" they would apply to states produced by having been struck by any agent, solid or gaseous, and the lesions of which are focal with bruising of tissues. The term "emotion" they apply to states due to exhaustion and over-exertion, purely functional conditions—no organic lesions.

They also call attention to the similarity in the nature and variety of the symptoms of the later lesions to those seen during the first days after a shock. The persisting sequelae and later complications are sufficiently frequent to darken the prognosis of shock even in the simplest cases. Their experiments showed that a lesion which may be simply vascular at the beginning of the disease, may be the starting point of a secondary, grave and important disability.

In considering the value of the above work, it must be remembered that it is not always possible to draw a comparison between effects of concussion on rabbits and human beings. The skeletal system of a rabbit is so frail when compared to that of man that it is necessary to take into account the force used in making experiments, and this is an extremely difficult matter to determine with any degree of scientific accuracy.

KESCHNER, New York.

LUMBAR PUNCTURE AS A FACTOR IN THE CAUSATION OF MENINGITIS. PAUL WEGEFORTH, M.D., Am. J. M. Sc. **158**:183 (Aug.) 1919.

During the past year the Army Neurological School has reported results of studies made on experimental meningitis. The report showed that lumbar puncture performed on animals which had been given an intravenous injection of an organism which possessed high pathogenicity for the meninges produced a fatal meningitis.

Wegeforth and Latham attempt to show the bearing of this work on lumbar puncture performed in human beings suffering from septicemia. They performed lumbar punctures on ninety-three patients who were suspected of having meningitis. A positive diagnosis of meningitis was made in thirty-eight instances at the first puncture, and fifty-five yielded spinal fluids which contained neither pus nor organisms. Among these were six cases which at the time of the puncture had yielded positive blood cultures, three being meningococcus and three pneumococcus septicemia. Five of these cases subsequently developed meningitis—one, a pneumococcus case, recovered.

They state there were four cases of pneumococcus meningitis, and that two of these cases developed a meningitis after the withdrawal of a clear normal fluid. One of them had rigidity of the neck, and was stuporous at

the time of the first puncture. Seven days later the second puncture yielded a negative fluid. Two days after this puncture there was definite rigidity of the neck and marked Kernig's sign, and lumbar puncture performed at this time yielded a cloudy fluid containing 1,200 leukocytes per c.mm., increased globulin per content, and the pneumococcus was present in both smear and culture.

The question that naturally arises in discussing a case of this kind is whether the meningitis was present at the time of the first puncture. It is noted that there was some rigidity of the neck—which proved that there was probably some irritation of the meninges at that time. If the clinical symptoms had not suggested the probability of a meningitis, lumbar puncture would not have been performed. A negative spinal fluid does not necessarily indicate the absence of an inflammatory process in the meninges. It has also been clearly established that there may be small microscopic foci of bacteria in the meninges which give no evidence of their presence either in the spinal fluid nor even macroscopically in the meninges after death, and that only careful microscopic examination of the meninges will reveal their presence. Liebermeister has stated that "in 25 per cent. of the cases of pneumonia there is distinct suppurative inflammation in the membranes of the spinal cord, even though their gross appearance is unchanged." Therefore, the case used by the authors as illustrative of the effect of puncture in producing meningitis is certainly not an unquestioned demonstration of their thesis. The second case cited was that of a soldier being treated for pneumonia who had severe headache and an atypical rash that turned out to be acne. The combination of symptoms was so suspicious that a surgeon on the meningitis service was called in consultation. The authors state that in order to be certain of his diagnosis, the specialist ordered a lumbar puncture. The fluid was clear. A blood culture was then taken and revealed the presence of pneumococcus. The following day the patient showed, what is stated to be unmistakable, but what I would consider were more definite signs of meningitis. A second lumbar puncture was performed, and this time the spinal fluid showed the presence of meningitis pneumococcus. This case likewise, therefore, is open to the same criticism that was made on the previous case. The authors look on these two cases as indicating the possibility of the removal of spinal fluid acting as a factor facilitating the infection of the meninges from the blood stream, and even go so far as to state that in the second case the obvious purpose was to "complete the clinical study," which is not borne out by the reasons which they themselves give for the puncture. As stated above, in neither of these cases can we be certain that the meningitic process had not already commenced in the meninges before the removal of the spinal fluid.

The authors describe a number of cases of meningococcus in which there were three of the fulminating type. These three cases were of very short duration—from fifteen to twenty-four hours. There were no symptoms indicative of irritation of the central nervous system, and yet the spinal puncture revealed the presence of meningitis. In these cases the severity of the septicemia unquestionably overshadowed entirely the symptoms of meningitis, and showed clearly that a meningitis causing very slight changes in the spinal fluid and with the presence of organisms in it may pass unnoticed. One of the cases is worthy of being cited in more detail. The patient was admitted to the hospital, Oct. 2, 1918, at 5:30 p. m., complaining of general malaise, pains in the back, legs and joints, headache and sore throat. He was conscious and showed no signs of meningeal involvement. The skin was covered with

ecchymotic areas. His temperature was 103 F.; pulse, 112. Blood culture demonstrated a meningococcus septicemia. Lumbar puncture performed at the same time yielded clear fluids—4 cells per c.mm. and there were no organisms in the cultures or spreads. The patient was given serum intravenously. On the morning of the following day he was markedly prostrated, but could be aroused. The neck was stiff, the reflexes were abolished. The patient died at 11:30 a. m. The pathologic examination showed the presence of an early meningitis affecting the cerebral hemispheres. The authors probably claim that this early meningitis is evidence of the action of the spinal puncture on the invasion of the meninges in a meningococcus septicemia. The weakness of such an argument lies in the fact that they cannot assert in the light of our present knowledge that this early meningitis was not present before the spinal puncture, even though no organisms were found in the fluid. In the second case they reported, that of a soldier, E. A., who died twenty-four hours after the onset of his illness, and in whom no lumbar puncture was performed during life, a specimen of fluid obtained three hours post-mortem revealed a colony of gram-negative diplococci among the contaminating organisms. They were not able to grow this subculture sufficiently to type it. No gross lesion of the central nervous system was seen, but the histologic finding of the meninges showed an infiltration with large mononuclear cells. In this case it is evident that there was a beginning meningitis, even though there were no clinical signs, and even though the fluid showed only one gram-negative coccus. Had this case been punctured as had the other two, and a negative fluid obtained, and had a subsequent puncture performed either a few hours or days later revealed a positive result, the authors would probably have claimed that the puncture was the cause of the invasion of the meninges, whereas the beginning of this meningitis was present probably shortly after the onset of the infection.

They also describe a case of septicemia which recovered without showing meningeal involvement. This patient had convalesced from pneumonia, and had had a normal temperature for five days, when it suddenly rose to 103 F., and a morbilliform rash appeared. A blood culture taken revealed a meningococcus septicemia, but twelve hours later the temperature and pulse were normal, and a second blood culture taken was sterile. Lumbar puncture was not performed, and the patient recovered. This case, of course, cannot be used as an argument against lumbar puncture, because there were no signs of meningitis present, and therefore despite the meningococcus septicemia, there was no indication for the performance of a lumbar puncture. The reviewer, however, thinks that had a lumbar puncture been performed, it would have made no difference in the course of the disease, since the patient's natural resistance to the meningococcus was evidently either extremely high, or the pathogenicity of the meningococcus was extremely low.

The authors report a case of meningococcus septicemia, showing no clinical signs of meningitis, in which a lumbar puncture was made after the patient had received serum treatment. The spinal fluid had a slight blood contamination, and a blood-agar plate showed a single colony of meningococcus. The patient did not develop meningitis. The authors consider the presence of the bacterial colony due to blood contamination, and yet they state that the meninges in this instance could hardly have escaped infection "for if the organism were not already present in the subarachnoid space at the time of the puncture, there was a possibility of indirect and direct inva-

sion from the blood stream." They account for the absence of the meningitis by assuming that the organisms had already lost their virulence, probably as a result of the intravenous serum therapy.

They cite two cases in which as soon as the clinical diagnosis of meningococcus septicemia was made serum was given intravenously, and within twelve hours specimens of blood and spinal fluid failed to show the presence of organisms. Both of these patients recovered without developing meningitis. Since no puncture was made before the intravenous injection was given, however, there is no way to determine whether or not the meninges were involved.

Two patients with meningococcus septicemia were punctured during the period of blood stream invasion, before serum was administered. The first was recovering from pneumonia, and had a normal temperature for sixteen days; it then rose to 103. He had slight headache and pain in his legs, but no other symptoms of possible meningeal involvement. The following day petechiae appeared and there was a slight Kernig's sign. Blood cultures at this time showed meningococcus, Type IV, and lumbar puncture revealed thirteen white cells and 600 red cells per c.mm. No organisms were obtained. Intravenous treatment was started and on the second day symptoms of meningeal irritation were marked, and the second lumbar puncture showed a cloudy fluid with numerous pus cells and many intracellular and extracellular gram-negative diplococci. Intraspinal and intravenous treatment was then instituted, and the patient made an uneventful recovery. The second case was similar to the first except that the patient died. Necropsy showed a widespread meningitis. The authors admit that in both of these cases there was some blood contamination of the spinal fluid at the first puncture, and admit the possibility of direct infection of the meninges through injury of the blood vessels by the exploring needle. They are, however, inclined to disregard this possibility, but we cannot overlook it in forming a fair judgment of the matter.

Herrick and Dannenberg (J. A. M. A. **73**:1322 [Nov. 1] 1919) described the results of their studies of the cerebrospinal fluid in acute disease, and stated in reference to lumbar puncture that "Of late, much has been said and written from a laboratory point of view of the dangers of lumbar puncture. After an experience with some 5,000 lumbar punctures in a large military medical center, in all sorts of medical conditions, we are not impressed with its clinical dangers. In none of the cases tabulated did true meningitis develop. Animal experiment has indicated possible dangers, under very special conditions, but its results cannot always be wisely translated bodily into terms of human pathology. Until convincing clinical proof is at hand, the skilled clinician need have no hesitation in removing from 5 to 8 c.c. of cerebrospinal fluid through a small needle, drop by drop."

In their conclusion, Wegeforth and Latham state that infection of the meninges occurs not infrequently following the release of normal spinal fluid following the infection of septicemia. They justify five of their observations with this contention. However, a close study of these five cases will convince any one that their conclusions are not justified. They recommend the following:

1. "That careful consideration be given the bacteriological study of the blood before such punctures are attempted." No one would deny the importance of such a study, not only before a lumbar puncture, but in any case where septicemia is suspected.

2. "That in acute diseases, in the absence of definite signs of irritation of the central nervous system, lumbar puncture should be avoided unless it is first conclusively shown that the blood stream is free of infection." No clinician would think of doing a lumbar puncture whether the blood is free from infection or not if there were no definite symptoms of irritation of the nervous system. Among definite symptoms, however, must be included headache, pain in the limbs and slight rigidity of the neck, which the authors do not seem to recognize as symptoms of possible meningeal irritation.

3. "That when the clinical symptoms are such as to render a lumbar puncture advisable minimal quantities of fluid should be withdrawn, sufficient only to permit necessary laboratory tests to be made." The reviewer agrees with this conclusion, not because he believes the withdrawal of large quantities of fluid would facilitate the invasion of the meninges, but because it is unnecessary.

4. "That small-bore needles should be utilized in performing the operation to prevent as much as possible subsequent leakage of spinal fluid into the surrounding tissues." This conclusion is justified, not because the leakage of spinal fluid into the surrounding tissues would cause bacterial invasion of the meninges or infection of the surrounding fluid, but because the chances of trauma to vessels and the possibility of puncture headache are lessened by the use of the small needle.

In criticizing this article, and also the work of the Neurological School, the reviewer wishes to lay stress on the harmful influence which they may exert in the treatment and diagnosis of diseases of the central nervous system. It has taken us a very long time to convince both the lay public and the medical profession that in lumbar puncture we have not only a very efficient and important means of diagnosis, but also a procedure, which, intelligently used, can be regarded as absolutely harmless. Unless the profession examines the work of these various authors critically, we fear that the opposite impression will be created and that we will be handicapped not only in using the method, but what is more important, there will also be a tendency not to use this important diagnostic measure when it is indicated and when the results obtained by it cannot be obtained in any other fashion.

In conclusion, I wish to quote the last sentence of an editorial article which appeared in *The Journal of the American Medical Association* of Sept. 27, 1919: "The precautions recommended by Wegeforth and Latham are of importance in acute general infection. Nevertheless, until we have better means of differentiating between the various forms of meningitis, it would not appear advisable to delay spinal puncture unduly."

STRAUSS, New York.

PHYSIOPATHIC CONTRACTURES OF PARETIC EXTREMITIES CAUSED BY CRANIOCEREBRAL WOUNDS. Considerations on the Pathogenesis of these "Reflex" Disturbances. MAJOR LIONELLO DE LISI, Riv. di patol. nerv. **24**: Pts. 1 and 2, March 25, 1919.

The author does not refer to the cases of cyanosed, hyperhydrotic, edematous extremities seen in the emotional war reactions, but to actual contractions. These are not like the ordinary hemiplegic contractural attitudes, but are accompanied by all the trophic-idiomuscular-vasomotor and reflex disturbances of true paralysis. The cases do not correspond to the "reflex disturbances" described by Babinski. The author recognizes the possibility

that in his cases, despite the fact that the actual paralysis is caused by craniocerebral wounds, the atypical contractures may be hysterical in origin. He has never seen a case in a simple hemiplegia due to brain injury. The contracture always develops in the upper extremity, and then only if there has been an injury of the extremity beside the craniocerebral injury. In the author's case illustrating these points, a soldier suffered a monoplegia (face and right upper extremity) from a fracture of the skull in the left parietal region on June 26, 1918. Contracture began ten days later. In April, the patient sustained a small wound on the dorsum of the right hand while cutting barbed wire. He considered the wound of no importance, and did not give the history until questioned concerning the scar found on examination. The oculocardiac reflex was inverted at first. The upper right extremity was held rigidly by the side, with the hand in the position of the obstetric hand, fingers all tensely extended and adducted, with the exception of the little finger and thumb, which were flexed strongly into the palm. The fingers could be forcibly moved, but gradually resumed the contractural attitude. There was atrophy of the entire extremity, including the small muscles of the hand. Electrical reactions were normal; the lower extremity was not paretic; there were no sensory changes, with the exception of diminished stereognostic sense in the affected hand; the deep reflexes were plus in the affected extremity with pronator clonus. There was no Babinski reflex. The abdominals and the other superficials, including corneal and pharyngeal, were normal. The affected hand was cold and cyanotic and then became sweaty; the same condition, but in a less degree, appeared in the left hand, especially in cold weather. The nails of the right hand grew rapidly, and were corrugated. The blood pressure was 135 in both extremities. The bones of the right hand showed osteoporosis.

The patient was mentally seclusive, apathetic, spoke little, was diffident and depressed. When stressed by examination or otherwise, he had tachycardia, sweating of the forehead, contractured hand and tremor. There was no change in this condition after the armistice. Only after many months of electrotherapy did the patient show any improvement.

The author differentiates the attitude in this patient's contracture by pointing out that it was caused by the small muscles of the hand rather than by the long extensors and flexors which cause the deformities in true or hysterical paralysis. In none of the author's cases were peripheral types of sensation disturbed. The most constant symptoms, besides the contracture, were the thermic and trophic changes in the extremity. Many authors have reported trophic and thermic symptoms in the extremities of true hemiplegics, even diminished secretion on the paralyzed side, as tested by pilocarpin. Some believe that the actual immobility in the paretic extremity, or the loss of energy in the destroyed cortex, causes toxins to be produced which travel along the same pathways as the trophic impulses to supramedullary centers, and thus cause the trophic and thermic symptoms.

Babinski's theory of paralysis of the sympathetic vasomotor control, causing reflexly diminished circulation, and therefore weakness and contractures in these cases, is refuted by the fact that in many cases where the vasomotor and contractural symptoms occur, no paralysis existed. An emotive psychologic factor may partly explain these cases.

Referring to his illustrative case, the author believes that without doubt the small wound in the right hand received in April, was the cause of the contracture which followed the craniocerebral injury, with monoplegia in

June; but this wound did not cause reflex disturbance until the extremity had become paralyzed, and then the contracture and other signs began ten days after the cerebral injury. He is inclined to believe that the emotional factor, besides the possibility of loss of cortical vasomotor control, must be considered. The factor of imitation, the patient having before him the picture of others afflicted like himself, is also noted. The author insists that his cases are not hysteria, although he admits that the vagus signs accompanying the emotions were present in his cases, namely, cold hands, hyperhidrosis, tachycardia, dermografia, occasionally low blood pressure, loss of weight, inversion of oculocardiac reflex, muscular hyperexcitability, tremor and even exophthalmos and Von Graefe's sign. In his cases these symptoms were explained by the acute and continuous emotional shock incident to fighting. Injury occurring at a time when the vasomotor mechanism is unstable, predisposes this mechanism, no matter what the injury and regardless of the degree of paralysis. The injury may, perhaps, by endocrino-sympathetic influences cause a defect in the nervous circulatory control, favoring an intoxication of the neuromuscular apparatus, with contractures.

OSNATO, New York.

FEVER IN HYSTERIA, WITH THE REPORT OF TWO CASES.

HUBERT S. HOWE, Neurol. Bull. **2**:137 (April) 1919.

While fever in hysteria is not at all rare, hysterical fever, that is fever of true psychogenic origin, is extremely uncommon. Its reported presence is usually looked on with suspicion, particularly so in view of recent studies on obscure focal infections. If one bears in mind the frequency of such infections and the difficulty of their detection, and the prevalence of hysteria, one can readily see how by the law of chance the two would occasionally be combined. The fact, therefore, that no cause can be found is no proof that none exists. However, too many cases have been reported by very reliable observers for one to doubt the existence of hysterical fever. Furthermore, we know that vasomotor changes can and do exist on a psychogenic basis, and theoretically we can well conceive of fever from psychic causes. So, too, the recent attempts to correlate hysteria with disturbances in the vegetative nervous system or changes in the endocrines, may help us to speculate on the possible origin of the fever. None the less, the warning cannot be made too strong not to attribute the fever to hysteria before all the possible sources have been exhaustively studied. Apparently Dr. Howe has done that in the two cases he has reported.

The first case was that of a young unmarried woman of 21. The only things worthy of note in her past history are an attack of appendicitis, for which she was operated, and swollen gums six months before the onset of the present illness. Vomiting, headache, fever and abdominal pains were the chief symptoms. The onset was sudden nine days before admission to the hospital. Physical examination was absolutely negative. The urine showed a faint trace of albumin on one occasion, but there were no casts or other pathologic findings. Blood culture, Widal's test, the test for malaria and the Wassermann test were negative. The spinal fluid showed nothing abnormal. The von Pirquet reaction was suggestive. The blood, on several examinations, gave a leukocyte count ranging from 4,000 to 10,200, the polymorphonuclears varying from 68 to 77 per cent. Cultures from the urethra were negative. Roentgen-ray examination of the chest was negative. The temperature varied

from 99 to 103 F., respirations, 18-22. During her stay in the hospital, from August 25 to October 9, she had repeated attacks of unconsciousness which showed all the earmarks of hysteria and none of that of an organic condition. Her vomiting persisted, and she was transferred to another hospital unimproved.

The second case was that of a young girl of 16. The chief complaints were fever and pain over the right cheek for one and a half years; attacks of asthma for fourteen years. The past history was not particularly significant except that she had a submucous nasal operation, incision of the right ear drum, and drainage of the ethmoidal cells and right antrum. On several occasions she was treated at St. Luke's Hospital, and once operated on for supposed tuberculous peritonitis. She had retention of urine, necessitating catheterization for weeks, her temperature ranged between 100 and 105 F., but no diagnosis was made and she was finally discharged with the label, essential fever.

General physical examination by Dr. Howe revealed little that was positive, except that the chest signs were those of asthma. The roentgenogram showed a shadow of both lungs, especially of the left apex. The blood count: white cells from 10,000 to 18,000 and polymorphonuclears from 69 to 85 per cent. Blood culture, the Widal, malaria, Wassermann, spinal fluid and urine tests were negative. The von Pirquet test was positive. A special neurologic examination revealed: hybrid gait, not significant of any organic condition; loss of touch, pain, temperature and posture senses over the entire right half of the body, including tongue, gums and external auditory meatus, except over right malar bone; and bilateral corneal and pharyngeal anesthesia. The sense of smell was absent on the right side. The field of vision was concentrically contracted. Taste was absent on the right half of the tongue.

Prolonged observation tended to confirm the diagnosis of hysteria, although her high temperature persistently continued.

As the author well points out, there was no parallelism in either case between the fever and the rates of pulse and respiration. There were no concomitant fever signs, such as malaise, thirst, pains, loss of appetite, etc. The patients were as unconscious of their fever as of all other hysterical signs and symptoms. Of particular note was the fact that there was no sign of hypermetabolism which is so significant of true fever, and although the second patient gave a history of almost constant fever for eighteen months, she showed no loss of weight. In neither case was there evidence of tampering with the thermometer on the part of the patients.

From the evidence submitted, one must concede that the author has proved the presence of hysterical fever in two patients.

WECHSLER, New York.

THE USE OF ORTHOPEDIC AND PROSTHETIC APPLIANCES IN THE LATE TREATMENT OF WAR DISABILITIES. A. B. LEMESURIER, M.D., Medical Quarterly, Department of Soldiers' Civil Reestablishment, Canada **1**:179 (July) 1919.

In an illustrated article, Dr. LeMesurier describes several standard appliances that have proved of value in Canada in treating the following types of disabilities: (1) nerve injuries; (2) disabilities of the joints; (3) disabilities of the feet, and (4) amputations.

The author observes that in nerve injuries the muscles require treatment by various appliances, and that the aim of this treatment is to attain one of three objects: (1) to prevent even momentary overstretching of the paralyzed muscles,

(2) to prevent or correct deformity caused by contraction of unparalyzed opposing muscles, or (3) to hold the limb in the best position for function. He describes briefly, with illustrations, the appliances used in treating patients with musculospiral, ulnar and sciatic lesions, these nerves being most frequently injured.

Two musculospiral splints are described, which are somewhat similar to those developed in our own army hospitals at Cape May and Staten Island, but they are not as simple in construction, nor do they appear to be as comfortable or practical. In each splint a separate thumb piece is necessary to extend the thumb, and the first phalanges remain unsupported. The splints, however, appear to be quite up to standard in the function of relaxing the extensors of the wrist. Extension of the hand is secured by pressure beneath the metacarpophalangeal joints by means of a hand piece, the other two points of pressure being the wrist and the forearm.

The appliance for ulnar paralysis used in Canada is considerably more complicated than that used in this country, but appears to carry out well the requirement of flexion of the metacarpophalangeal joints (thus compensating for the unopposed action of the extensor communis digitorum). A gutter-shaped piece, in which each finger lies, is designed to produce extension of the interphalangeal joints. Such a device would be perfectly satisfactory when contractures producing marked flexion of the fingers do not occur, but some modification would probably have to be made in cases with severe fibrotic changes.

The external portion of the sciatic nerve is found to be the source of paralysis more frequently than the internal, even when the injury is above the bifurcation. The resulting foot-drop is treated at night by the use of a simple canvas and wire "L" shaped appliance to prevent the weight of the bed covers from causing plantar flexion of the foot. The author favors the use, during the day, of a steel brace, jointed opposite the ankle, with a stop which locks when 90 degrees is reached, but allowing free movement of the joint up to that angle. The brace is attached to the sole of the shoe with rivets. In paralysis involving the muscles supplied by the internal popliteal nerve, the author advocates the use of a similar brace with the ankle joint completely locked. This brace has much to recommend it, but on the neurosurgical services at Cape May and Fox Hills a small, light spring splint, which exerts a constant pressure upward on the sole of the shoe, was preferred. The author condemns the use of such splints because a certain amount of stretching of the paralyzed muscles must necessarily occur. Our experience led us to believe that complete prolonged immobilization of any joint is likely to lead to fibrous changes of a most undesirable character. It was believed that the degree of stretching permitted by the spring splint for foot-drop is not sufficient to be injurious, and is more than compensated for by the lightness, comfort and simplicity of the spring. In cases of injury involving only the internal popliteal nerve, in contradistinction to the author's practice of complete immobilization of the ankle joint, no mechanical appliances to prevent dorsiflexion were used in our neurosurgical centers.

In the remainder of the article are described: 1. Braces which may be worn by patients having flail elbow or knee joints with poor muscular control. (2) Appliances to be used when there is stiffness of the knee or elbow joint, short of bony ankylosis, where it is desired to increase movement. 3. Appliances for treatment of disabilities of the feet. 4. Prosthetic appliances to be used following amputations.

ARNETT, Philadelphia.

EPIDEMIC ENCEPHALITIS. A PRELIMINARY CONSIDERATION OF SOME OF ITS PROMINENT CLINICAL AND PATHOLOGICAL MANIFESTATIONS. FREDERICK TILNEY and HENRY ALSOP RILEY, Neurol. Bull. **2**:106 (March) 1919.

The authors describe twenty cases of epidemic encephalitis which came under their observation in 1918. While they consider the disease an entity of unknown pathogenicity with a distinct symptomatology, they enumerate eight, and possibly nine well-defined types, namely: lethargic, cataleptic, paralysis agitans, polioencephalitic, anterior poliomyelitic, posterior poliomyelitic, epileptomaniacal, acute psychotic and infantile (or neonatorum).

The lethargic type (four cases) represents the average picture which has so frequently appeared during the epidemic of 1918 and the present year. One case came to necropsy and only the medulla, pons, mid-brain and basal ganglions showed pathologic changes, most marked in the latter. Characteristic were pial injection and small hemorrhages visible on macrotomic section Microscopically there were revealed: small hemorrhages, engorgement of vessels, round cell infiltration, mild gliosis and a few ameboid glia cells, chromatolysis and cloudy swelling, very marked in the motor cells of the affected motor cranial nuclei, some vacuolization and slight neuronophagia. Although all the changes mentioned are seen in acute poliomyelitis, their character, intensity and variety were so distinct that one could not mistake the picture.

The cataleptic type is represented by two cases, both in young girls. Both showed marked lethargy and cranial nerve palsies in addition to the "image" rigidity characteristic of catalepsy. The paralysis agitans type includes five cases of acute onset. Two cases with cranial nerve involvement but no other symptoms are designated as the polioencephalitic type. One case of typical poliomyelitis anterior with prolonged somnolence, and one of poliomyelitis posterior with objective and subjective sensory involvement of the trigeminus and second, third and fourth sacral dermatomes together with lethargy and ophthalmoplegia, represent types 5 and 6. Characteristic of the epileptomaniacal type is a patient whose illness began with hallucinosis and delusions, followed by prolonged lethargy and later by maniacal excitement. Finally, for two weeks before death, epileptiform seizures occurred several times a day, culminating in a terminal status epilepticus. Strabismus and nystagmus showed involvement of the cranial nerves. One case showed an acute psychosis with hallucinations, delusions, disorientation, etc., on top of the lethargy. Finally three cases are described in infants, from 4 to 6 weeks old, who suddenly passed into prolonged somnolence during which they developed cranial nerve palsies.

In a critical analysis of the symptoms, the authors emphasize the comparative suddenness of onset, the profound stupor, the prevalence of cranial nerve palsies, the presence of some degree of fever, the tremor, particularly of the paralysis agitans type, the profound asthenia, restlessness and occasional hypertonus. They do not consider the spinal fluid of pathologic significance. This is rather at variance with the findings of other observers who claim that a pleocytosis even up to 200 cells is frequently encountered. The prognosis is looked on as rather grave, the authors' series having given a mortality of 25 per cent. It is worth mentioning that the epidemic of 1919 (and it seems to be quite as prevalent as that of 1918) is very much milder, the reviewer not having seen one fatality in more than twenty cases.

The authors further speculate on the nature of the disease and carefully seek to distinguish it from the poliomyelitic group or the influenza epidemic,

although they were not fortunate in bringing direct evidence to bear on the specificity of epidemic encephalitis. They evidently were justified in their speculations, as Strauss and Loewe (J. A. M. A. **73**:1056 [Oct. 4] 1919) have successfully isolated and cultivated an organism from nasal washings, throat cultures and the spinal fluid of patients suffering from epidemic encephalitis, transmitted the disease to rabbits, obtained it from the animals and passed it through several generations.

The very thorough description by Tilney and Riley of the various "types" of epidemic encephalitis, deserves special consideration. They have also mentioned, from a review of the literature, a number of other "types," such as polyneuritic, cerebellar ataxic, hemiplegic, diplegic, etc. While this testifies to their accuracy of observation it appears to the reviewer that it is not wise to multiply "types." The important question ultimately is: Are we dealing with a specific disease? If we are, and there seems to be no doubt about it, then the various manifestations no more deserve to be called "types" than a tumor of the brain, say a glioma, should be variously designated because it happens to take root in different parts of the brain. Obviously the symptoms or "types" or "syndromes" vary with the location of the tumor, and so will the manifestations of epidemic encephalitis depend on the site of the lesion.

But while there is justifiable fear of possible confusion from the creation of types out of a specific pathologic entity, their careful description deserves attention for another reason. The correlation of symptoms with the various sites of the lesions serves definitely to establish the functions of those parts of the brain which are as yet not clearly understood. Our knowledge of the physiology of the brain will be greatly enhanced if we shall be able to show that a pathologic lesion has given a certain symptom or group of symptoms. So, too, will careful observation further our knowledge of pathology, and one need merely allude to the acute Parkinsons which point to the possible genesis of chronic agitans.

WECHSLER, New York.

SOME BIOLOGICAL EFFECTS DUE TO HIGH EXPLOSIVES. ALFRED CARVER and A. DINSLEY, Brain **42**:113, Part 2, 1919.

Admitting the importance of the psychogenic factors in the production of war neuroses, the authors of this article believe that a certain proportion of the nervous symptoms observed in soldiers after exposure to high explosives are physiogenic, or mechanical, in origin. In order to secure evidence to support this view, a series of experiments were made on animals by subjecting them to the effects of high explosives. A brief description is given of the properties and effects of explosives. An interesting fact presented is that high explosives may be sensitized, or made more easily detonated, by being repeatedly subjected to detonations insufficient to explode them. The inference is drawn that the nervous system may be affected in a somewhat analogous manner, resulting in a neurosis based on mechanical more than on emotional factors. Depending on the results produced at different distances from the point of detonation, three zones are roughly distinguished: zone A, or the "zone of brissance," is delimited by the area of gross disruption; zone B, the zone of decompression, and zone C in which the effects of detonation are further modified. Two series of experiments on animals are described, one on fish by exploding submerged charges of explosives, and one on rats and mice in cages at different distances from the explosives. In both series of experiments the animals as in zone A, were killed by the disruptive force. In

zone B most of them were stunned but eventually recovered. In zone C the effects were less marked, but excitement and irritability were noted. The mammals that were repeatedly exposed in this zone became stuporous. Dissections showed lacerations and hemorrhages in the animals which were killed, and capillary engorgement in those surviving. No histologic examinations were made. Some interesting observations are made of healthy soldiers in an "ammunition proof and demolition section" during the course of their ordinary duties. They were well sheltered, and the element of fear was minimal. Various symptoms were noted, including active tremors, increased pulse and respiration, and in a few cases, vomiting. Those most affected became less resistant to subsequent exposure. As a result of their experiments and of observations on soldiers, the authors believe that the "indirect concussion" of high explosives produces definite changes in the central nervous system, accounting for certain of the symptoms usually ascribed to psychogenic factors. Although they present no histologic evidence to support this view, their argument is very suggestive, and in line with other evidence of the same nature.

INGHAM, Philadelphia

SUBTEMPORAL DECOMPRESSION FOR ACROMEGALY; PRESENTATION OF A CASE. S. P. GOODHART, Neurol. Bull. **2**:168 (April) 1919.

In the words of the author, the patient was shown "for the purpose of demonstrating the value of subtemporal decompression in certain cases of acromegaly in which pain becomes persistent and intractable, etc. It involves furthermore consideration of the indications for either simple decompression, or a procedure of more radical nature, sellar decompression, with or without effort at removal of soft tissue."

The patient's illness had lasted several years. It began with frontal and occipital headache, pains later radiating to the face and shoulders. Later there developed weakness, loss of 40 pounds in weight, polydipsia, polyuria, glycosuria and diminished libido and potency. The bony changes in the face and extremities established the diagnosis of acromegaly. Syphilis was also diagnosed, for which the patient received antisyphilitic treatment. He was bedridden because of the severe pain; opotherapy had no effect. Spinal puncture gave no relief. Right subtemporal decompression five years after the onset of the illness resulted in complete disappearance of the pain. Immediately after the operation a temporary left homonymous hemianopsia developed; later the left pupil became dilated and reacted sluggishly to light. Left ptosis and weakness of the left internal rectus developed, and a few days later a bitemporal hemianopsia. These conditions were transitory, and were explained by the author on the supposition of an intraventricular edema of the third ventricle. This is rather problematic and would hardly explain all the signs. However, the fact remains that all of the patient's symptoms cleared up after the operation, though the bony changes remained uninfluenced.

In discussing the case, the author explains how difficult it is to make a diagnosis of struma of the pituitary or enlargement of the sella despite symptoms of acromegaly or dystrophia adiposis genitalia, and points to the unreliability of the roentgen ray in determining the size of the sella. The last point is particularly well taken, and the author justly urges the frequent making of roentgenograms before making a definite statement as to the size of the sella. It may be added that stereoscopic plates should never be omitted. In this connection one may recall an article by Howe (Neurol. Bull.) con-

taining a study and measurements of forty sellae in normal people, and showing such wide variations that one can hardly distinguish some of them from sellae abnormally enlarged. It should be added that a great deal depends on the angle at which a picture is taken. As to the difficulty of making a diagnosis of struma of the pituitary, Goodhart describes briefly a typical case of Froehlich's syndrome which on necropsy showed a tumor of the corpora albicantia. He further dilates on the frequent presence of marked pituitary symptoms without macroscopic changes, and points out that subjective symptoms may be due to a "temporary true hyperplasia of the whole gland with resulting distention of the sensitive dural envelop, or to a local edema. In the absence of objective evidence of a neoplasm or strumous growth within the sella, it would seem that for the relief of the persistent and agonizing pain the comparatively harmless surgical procedure of the subtemporal decompression should be utilized."

While there is no great objection to a subtemporal decompression (although the operation is not altogether harmless), and it certainly is preferable to a direct attack on the sella, wherever it can possibly be avoided, it is rather difficult to decide when the lesser operation is indicated, unless at least some signs of intracranial pressure accompanying the pituitary symptoms are found. However, the fact remains that Goodhart's patient obtained marked relief, and his suggestion certainly is worth serious consideration.

WECHSLER, New York.

GUNSHOT WOUNDS OF THE SCALP, WITH SPECIAL REFERENCE TO THE NEUROLOGICAL SIGNS PRESENTED. GEOFFREY JEFFERSON, Brain **42**:93, Part 2, 1919.

The author, who served as surgical specialist attached to the Fourteenth General Hospital, British Expeditionary Forces, presents a report of fifty-four cases of unselected wounds of the scalp without cranial fracture, calling particular attention to the symptoms indicating injury to the brain. Of the fifty-four cases, forty-nine presented early neurologic symptoms which were classified into two types, namely, those in which there were signs of general disturbance and concussion, and those showing evidences of definite local cerebral contusion. The general symptoms, or those due to concussion, occurred in practically all of the cases, and may be summarized as follows: disturbances of consciousness; transient unconsciousness in 25 per cent., and a dazed or stunned condition in 25 per cent. Headache was noted in 82 per cent., most frequently frontal, and was "fixed," or of maximum intensity near the wound, in 45 per cent. of the cases. Giddiness was common, but was only noted on gross changes of posture. Nausea and vomiting were less frequent, vomiting occurring in eight and nausea in thirteen cases. Tendon reflexes were increased in twenty cases, eight of which included increased arm jerks which indicated more serious cerebral injury. Local cerebral contusion was indicated when focal symptoms were present, and occurred in seventeen cases (thirteen motor and four visual). The severity of the symptoms varied from a slight unilateral increase of the tendon reflexes to a definite hemiparesis and jacksonian convulsions. Contralateral cerebral contusions were noted in four cases, all in the motor area. In most of the cases the symptoms, both general and focal, were of a transient character, but in a few slight residual symptoms remained after several months. Some of these late symptoms were ascribed to a secondary hysteria, but the evidence presented to sustain that view is superficial

and unconvincing. Three of the patients of the series were trephined, in two of whom extradural hemorrhages were found in the absence of cranial fracture.

INGHAM, Philadelphia.

MENTAL AND NERVOUS CHANGES IN THE CHILDREN OF THE VOLKSSCHULEN OF TRIER, GERMANY, CAUSED BY MALNUTRITION. SMILEY BLANTON, Mental Hygiene **3**:343 (July) 1919.

Blanton presents the result of a study of the schoolchildren of Trier, one of the German Rhenish province cities, made by the department of Sanitation and U. S. Public Health Service. Six thousand five hundred pupils were surveyed. They were between the ages of 5½ and 14, and for three years they had been under the disadvantage of an inadequate diet (particularly deficient in animal proteins, butter fats and milk). Special interest attaches to the psychologic-psychiatric findings. Retardation, by which the author understands a failure to pass one or more grades, increased 7 per cent. in the "war" years 1914-1918. It is estimated that 7.39 per cent. of the entire school population was retarded primarily by reason of malnutrition. In view of the fact that the usual promotion standards were much relaxed, this estimate is probably low. Apparently neither outspoken feeblemindedness (0.95 per cent.) nor the group including "abnormal," "nervous," conduct disorder, organic nervous diseases, neuroses and psychoses (fifty-four cases) were affected by the nutritive enfeeblement. That only two cases of neuroses are reported is rather surprising, as Trier was bombed twenty-two times and there were ninety-seven "alarms." Only 5 per cent. of the children showed speech defects although minor difficulties such as slurring, indistinctness and lisping were exceedingly common in the younger children, indicating retarded speech development which might fairly be attributed to malnutrition. Increased fatigability, inattention, slowing of comprehension, poor memory and restlessness were almost universal.

This survey is open to the same objections attending all "large scale" studies. The practical impossibility of accurate individual examination and observation, and the necessity of conducting the work through an interpreter, must be given some weight in a consideration of the findings. However, with some allowance for the possible margin of error, the results furnish a valuable indication of the effects of malnutrition on the mental health and growth of schoolchildren.

STRECKER, Philadelphia.

PARALYSIS OF THE GLOSSOPHARYNGEAL, PNEUMOGASTRIC AND SPINAL ACCESSORY NERVES WITH CEREBELLAR SYMPTOMS. W. G. SPILLER, J. Nerv. & Ment. Dis. **49**:481 (June) 1919.

Spiller reports the case of a man of 45 years of age who gradually developed lateral movements of head, difficulty in swallowing with a tendency to choke, vertigo, slow speech, lateral nystagmus, difficulty in protrusion of tongue, anesthesia of uvula and soft palate with paralysis of the palatopharyngeal muscles, but no impairment of the functions of the vocal cords. There were in addition evidences of a lesion of the internal ears with the Bárány test; intention tremor of each hand, adiadochocinesis, loss of tendon reflexes in lower limbs with incoordination of gait; the blood Wassermann test was positive, but the cerebrospinal fluid findings were negative.

These symptoms pointed to cerebellar disease and also to paralysis of the glossopharyngeal and pneumogastric nerves probably due to a syphilitic meningitis implicating these nerves at their exit from the medulla oblongata.

It would appear from the paralysis of these nerves and the integrity of the vocal cords that there is a distinct nuclear representation of the motor supply of the soft palate and larynx, and that the former receives its motor supply from the spinal accessory and the sensory supply from the pneumogastric. Spiller calls attention to the rare occurrence of isolated paralysis of the glossopharyngeal and pneumogastric portion of the spinal accessory.

YASKIN, Philadelphia.

POSTURAL TONE OF THE RECTUM, WITH SPECIAL REFERENCE TO DYSCHEZIA (RECTAL CONSTIPATION). A. F. HURST, Seale Hayne Neurological Studies **1**:208 (April) 1919.

Hurst contributes an interesting experimental study of dyschezia (rectal constipation) from the standpoint of rectal postural tone. By means of the introduction into the colon and rectum of rubber balloons with pump and manometer attachment, the author was able to determine the pressure (in millimeters of mercury) required to produce the sensation of rectal fulness and the call to defecation. Ordinarily this occurs when the peristaltic passage of feces from the pelvic colon is reflexly excited, by taking food into an empty stomach, drinking a glass of cold water, exercise, etc. The practical deduction from Hurst's work is a logical explanation of rectal constipation, on the basis of relaxed postural tone in the muscular wall of the rectum, which results when for any reason the call to defecation is habitually neglected. Then the sequence of events is as follows: Feces accumulate in the rectum; the muscular coat becomes more relaxed and, due to the diminishing postural tone, more expulsive effort is required. The relaxed muscular coat is inadequate and therefore evacuation is never complete; feces are always present in the rectum which normally should be clear. The lumen becomes permanently enlarged; the postural tone continuously decreased; obstinate constipation and, in extreme cases, complete muscular atony results. The author's work is in accord with Sherrington's observations and extends a very fruitful field of study.

STRECKER, Philadelphia.

A PATHOLOGIC REFLEX OF THE GREAT TOE: THE REFLEX OF THE SECOND PHALANX. PIERS BOVERI, J. Nerv. & Ment. Dis. **49**:385 (May) 1919.

Boveri presents a new sign consisting in flexion of the second phalanx of the great toe and obtained by striking the tendo achillis while the patient is in a prone position with the legs flexed at right angles to the thighs, so that the lower limbs form, by the angle of the feet and legs, the position of "Z." In normal persons there is flexion of the feet, but not of the phalanges. The pathologic sign, which is usually accompanied by loss of the Achilles jerks, is observed in lesions of the sciatic nerve, of the roots and of the spinal cord. The author cites ten cases in which the sign was observed. Boveri concludes that this sign is diagnostic of a partial lesion of the great sciatic, of the external popliteal or of their corresponding segments in the cord. The author does not offer any definite mechanism of the production of the reflex, but suggests as possibilities that it is either a result of reflection (?) or a "propaga-

tion of the stimulus which, not normally developing for the interruption of the superficial muscles of the calf, runs more deeply into the big toe."

The explanation of the cause as furnished by the author is not quite clear. There may, however, be some value to this reflex.

YASKIN, Philadelphia.

QUALIFICATIONS OF THE PSYCHIATRIC SOCIAL WORKERS. JESSIE TAFT, Mental Hygiene **3**:427 (July) 1919.

Taft describes the ideal psychiatric worker as one who has at her command a "working psychology which will be able to carry out in social treatment the psychiatrist's interpretation of a patient, so far as it can be done in terms of personality." This worker cannot be defined either in terms of college education or mature years, for neither necessarily guarantees the intellectual and emotional maturity which are essential. Experience in social service is not a requisite, provided there is some familiarity with simple case work. The candidate must not be too introspective about her own mental adjustment nor should she be so extroverted as to be unsophisticated in respect to the ordinary social-psychiatric problems of the community. The born worker in this field will be "a maternal sort of a person even if she is only 20." She will have worked out a satisfactory philosophy of living and her basic attitude toward life will be one of sober optimism. She will have confidence, poise, sympathetic understanding and mental flexibility.

STRECKER, Philadelphia.

EXPERIENCES IN THE IMMEDIATE TREATMENT OF WAR NEUROSES. EDWARD A. STRECKER, Am. J. of Insan. **76**:1 (July) 1919.

This is a readable and concrete account of the neuroses of the 28th division observed during the first hours or days by a psychiatrist who had to get results. The treatment consisted of hot drinks, good meals, optimism, appeal to patriotism, explanation; "trick cures" and hypnotism were avoided. This scheme was efficacious; it sent most of the men back to duty within four days, and left no damage.

Strecker concludes that to cure neuroses in civil life, to make a business of getting results, demands the removal of legal shackles and public indifference, a comprehensive plan and a new attitude of prognostic faith on the part of the physician.

BOND, Philadelphia.

POSTURAL LENGTH AND POSTURAL TONE IN HYSTERICAL AND OTHER DEFORMITIES. A. F. HURST, Seale Hayne Neurological Studies **1**:203 (April) 1919.

Hurst makes the very instructive observation that abnormal postures produced by hysterical contractures persist after consciousness is lost in sleep or even in deep anesthesia, notwithstanding the fact that the contracture itself disappears. He believes that the abnormal position is due to a change in the postural length and tone of the muscles, and is independent of the hysteria. The argument is convincing. By the normal postural length and tone of a muscle fiber at rest is understood a position about halfway between its extreme contraction and relaxation lengths. With Sherrington's work in mind, it is easy to conceive how a contracted position maintained for some time, might readily temporarily shorten the postural length, after the contraction has been

removed. An argument against the hysterical nature of such shortening lies in the fact that it is only to be corrected by voluntary and conscious reeducation on the part of the patient and is unaffected by psychotherapy. If this theory holds true, it will no longer be reasonable to regard the abnormal positions which sometimes persist as sequels to recovered organic nerve lesions, as hysterical additions. The conception also has an application to the field of orthopedics, and the normal postural length may have some weight in deciding on the best immobilizing position for a disabled limb.

STRECKER, Philadelphia.

HYSTERICAL TRISMUS AND OTHER NEUROSES OF THE JAW. S. H. WILKINSON, Seale Hayne Neurological Studies **1**:196 (April) 1919.

Wilkinson presents an interesting group of jaw neuroses. He feels that both autosuggestion and heterosuggestion play an important part in the development of the symptoms and that various jaw disabilities which have origin in actual disease and injury, may in this way be perpetuated as hysterical symptoms. Four cases of persistent jaw tremor (one having a duration of two years), three of trismus and one each of clonic spasm and palsy are described. The ease and rapidity with which the symptoms were removed bespeak the author's expertness with psychotherapeutic technic. Exception might be taken to the statement that under powerful suggestion "quite normal people may become the victims of hysterical symptoms." Symptoms of the type shown by the author's patient, would seem to imply the existence of an hysterical make-up, and it is difficult to conceive that this may be compatible with normality.

STRECKER, Philadelphia.

THE EDUCATION OF AN EPILEPTIC. E. PONCE RODRIGUEZ, Rev. de Psiquiatria **1**: No. 2 (October) 1918.

The author, a pedagogue and not a physician, states his theory of the service that can be rendered to an epileptic by intensive training. This service should be rendered under the supervision of the physician in charge of the case.

He reports the case of an epileptic boy, 10 years of age. He had had attacks since infancy, and was markedly defective. This was shown especially in the lack of a vocabulary; he was able to say only a few words. His attention wandered and could be held on one subject for only a few seconds. In the motor sphere there was great activity — he threw things about impulsively. In addition, there was a certain functional deficiency in the use of the hands, which was evidenced by a lack of synergic control rather than by a paresis.

The author began his treatment slowly with simple games and amusements, attempting first to train the attention, then to increase the words in the boy's vocabulary, and finally to inspire ideas.

After three years of "assiduous labor," there was considerable improvement. The vocabulary had become "notably enriched"; the attention, except before an attack, was fairly stable, and the association of ideas seemed fairly normal. The defective life was favorably influenced. He showed interest, and became enthusiastic about music.

The frequency of the epileptic crises was markedly diminished. Instead of grand attacks, appeared equivalents and frustrated attacks.

In conclusion, the author states that this type of treatment—the care taken by the pedagogue—has great advantage in that the patient, instead of becoming a chronic invalid in the hands of a well-intentioned but not understanding family, is able to develop to a large extent and become more capable of self control.

SOLOMON, Boston.

THE BASAL METABOLISM IN HYPOTHYROIDISM. J. H. MEANS, M.D., and J. C. AUB, M.D., Arch. Int. Med. **24**:404 (Oct.) 1919.

This article comprises the observation on six patients, and includes practically every clinical condition associated with hypothyroidism. The methods employed consisted briefly in the determination of the gas exchange of the recumbent subject, while in the postabsorptive condition, by means of the Benedict apparatus and the calculation of the heat production therefrom. The heat production is calculated from the oxygen absorption and the respiratory quotient obtained, with few exceptions.

The existence of lowered respiratory metabolism in hypothyroidism and the ability of the thyroid gland ingested to raise it has long been known. The metabolic rise may range anywhere from 15 to 72 per cent. The percentage of basal metabolism in hypothyroidism varies, as low as 33 per cent. below the normal for age and sexes.

The authors claim that the determination of the basal metabolism forms a sound and convenient method for governing the dose of thyroid preparation in cases of hypothyroidism far better than the clinical picture and is of value in differential diagnosis, and that the proper dosage can be far more accurately gaged by following the effect on metabolism. The aim should be to bring the patient to the normal level (about 3 to 4 grains daily) and then to find the minimum dose that will be effective (about 1 to 2 grains).

DELONG, Philadelphia.

Society Transactions

THE AMERICAN NEUROLOGICAL ASSOCIATION

Forty-Fifth Annual Meeting at Atlantic City, N. J., June 16-18, 1919

JAMES H. MCBRIDE, M.D., *President*

1. PRESIDENTIAL ADDRESS. DR. JAMES H. MCBRIDE. This article appeared in the August issue, p. 149.

2. BRAIN SURGERY IN THE WAR. DR. HARVEY CUSHING, no discussion. This article appeared in the November issue, p. 493.

3. PROBLEMS IN THE SURGICAL TREATMENT OF INJURIES TO THE PERIPHERAL NERVES: THE OUTLOOK FOR THE FUTURE. DR. CHARLES A. ELSBERG and ANDREW H. WOODS. This article appeared in the December issue, p. 645.

DISCUSSION

DR. GEORGE E. PRICE: I was very glad to hear Dr. Elsberg say that he placed no dependence on Tinel's sign. In our work overseas Tinel's sign proved so unreliable that it was discarded as being of no practical value and at Fort McHenry, where there was more opportunity to study the sign, we came to the same conclusion. Tinel's sign has been found in cases of complete interruption of a nerve with a wide bridge of scar tissue where one would not expect to find any nerve fibers. Even after recent complete section of the nerve, Tinel's sign has been present. Of the two theories advanced in explanation by Dr. Elsberg, one of the passage of nerve fibers through the scar tissue and the other of compensation of overlapping of sensation from other nerves, I would be in favor of the last. We have an example of that occurring normally in the musculospiral. An interesting motor problem is the inability to abduct the fingers in musculospiral palsy. If, however, extension is artificially supplied by placing the hand palm down on the table, the patient is then able to perform the movements of adduction and abduction of the fingers.

Dr. Elsberg also spoke of finding two cases of complete nerve section without loss of sensation, in the musculospiral and the internal popliteal. In the cases seen overseas and at Fort McHenry, there was no instance of total section of these nerves with complete preservation of sensation. In musculospiral cases there is usually a narrow band of anesthesia on the dorsum of the thumb that is apt to be overlooked.

One thing that the war has brought out is a marked improvement in the technic of neurosurgery, particularly in suturing operations on the nerves. In this connection it is only fair to say that some of the best results I have seen have resulted from operations by men who were neither neurosurgeons nor orthopedists, but general surgeons doing the best they could under adverse conditions.

DR. E. SACHS: This is very interesting work that Dr. Elsberg has done on the anatomy of the peripheral nerves. Similar work was reported some years ago by Stoffel who emphasized the importance of keeping this point in

mind in suturing the nerves. If one studies microscopically the regeneration of a nerve and notices the devious way a nerve filament grows, one cannot help feeling that it is not so very important, in suturing the two ends of the nerve, to attempt to approximate the proximal and distal end so very carefully. The principal factor in the successful results of peripheral nerve suture is the avoidance, as far as possible, of anything that may contribute to form scar tissue between the two ends. Dean Lewis's experiments showed how far a nerve might grow out if there was no obstruction between the two ends. This, I think, is further evidence that surgeons should avoid anything that may contribute to the formation of scar tissue and the approximation of the nerve ends.

Dr. Charles A. Elsberg: All I meant to say is that it seems to me there cannot be any question that, if a nerve has a certain structure and internal pattern, the best result will be obtained by bringing that nerve with all its fibers as near as possible into the anatomic relation in which it was originally when normal. *That* should be the ideal surgery. To be sure, nature is very good and she will cause the nerve fibers to grow around all kinds of obstruction, but that is no reason why the real object of an operation should not be the bringing of the nerve into a condition as near as possible to that which existed before the injury occurred. Any argument against such a procedure, tending to show excellent results when the nerve ends were sewed together haphazard, is not evidence of the successful work of the surgeon but of the successful work of nature. The surgeon should always suture the nerve so that its ends approximate as closely as possible to the normal nerve pattern, and the closer he gets to the normal condition, the better. *That* fact should be kept clearly before us. We can expect nature to help us, but we should make conditions such that nature can help us to the maximum.

4. GUNSHOT WOUNDS OF THE HEAD AND THEIR TREATMENT. Charles H. Frazier, M.D., and S. D. Ingham, M.D. This article appears in this number, p. 17. No discussion.

5. INFECTIVE NEURONITIS AMONG TROOPS ON ACTIVE SERVICE. Foster Kennedy, M.D. This article appeared in the December issue, p. 621. No discussion.

6. ACUTE ASCENDING PARALYSIS AMONG TROOPS, WITH PATHOLOGIC FINDINGS. Louis Casamajor, M.D. This article appeared in the December issue, p. 605. No discussion.

7. EXPERIMENTAL HEMATOGENOUS MENINGITIS. James B. Ayer, M.D. No discussion.

8. SENSORY CHANGES IN LESIONS OF THE MUSCULOSPIRAL NERVE. Arthur S. Hamilton, M.D. This article is a study of the sensory changes observed in cases of injury of the musculospiral nerve seen in the neurosurgical service of the Walter Reed General Hospital, referring particularly to the area of sensory involvement as compared with the anatomic distribution of the nerve.

DISCUSSION

Dr. Lewis J. Pollock: The anatomic distribution of the sensory fibers of peripheral nerves and the extent of sensory changes following section of peripheral nerves do not coincide. The area of analgesia in lesions of peripheral nerves has for many years been known to involve an area much

smaller than the sensory fibers of that nerve are believed to physiologically subserve. This has been explained on the basis of collateral innervation through adjacent nerves. The dissimilarity of sensory loss and physiologic nerve supply had been noted by Weir Mitchell who, in 1864, stated that it was a phenomenon not at that time explainable, but which showed that the knowledge which was anatomic in nature could not be used in clinical interpretations.

Recognizing the fact that the small area of analgesia and at times its total absence, as in cases of complete section of the musculospiral nerve, is due to the overlap of other nerves, it is apparent that the complete sensory distribution of a peripheral nerve is not only to the part of the skin which it physiologically subserves, but also to that part of the skin physiologically subserved by other nerves, which it, under certain conditions, supplies with some forms of sensation. Therefore, the total sensory distribution of a nerve is the physiologic distribution, plus its overlap.

It was with this problem that we concerned ourselves and the results were: For pain the overlap of the musculospiral nerve extends over the palmar surface of the thumb and the radial border of the palm to an extension of the line between the index and ring finger. On the dorsal surface of the hand, it extends to a line parallel to the radial surface of the little finger, and encroaches on the base of the ring finger. One-half phalanges of the index finger and one phalanx of the ring finger were supplied by the nerve. The overlap to temperature is somewhat less than this and to touch markedly less, so that on the palmar surface of the hand only a small border of the thumb is subserved by this nerve. The median, ulnar and musculocutaneous in the hand and the external popliteal in the leg, likewise, have considerable overlap.

The method by which this overlap was determined was: Complete lesions of the nerves innervating the hand were studied for the smallest area of the various types of sensory function which may be lost. From this study it could be determined that the remaining area of the so-called physiologic distribution of that nerve was supplied by the overlapping of other nerves and furnished an index to the limit of such supply.

If the hand is supplied by four nerves and two of these are out, it is obvious that the remaining portion of the skin in which sensation is intact must receive that sensation from the remaining two nerves. If, therefore, in a complete ulnar and median lesion, the interior surface of the hand in its radial half can feel pain, this pain must be subserved by the radial and musculocutaneous nerves. It remains only to study a combined lesion of the radial and median nerve when the extent of the musculocutaneous overlap is determined. What remains, therefore, in the various combinations of peripheral nerve lesions, certified at operation, such as ulnar and median, median and radial, ulnar, median, and radial, musculocutaneous, internal cutaneous and ulnar, etc., the complete sensory distribution of any peripheral nerve of the hand, may be determined by noting the residual sensibility.

The striking feature of this investigation is the large extent of skin which has been found to be subserved by the overlapping innervation of other nerves.

That this overlap subserves only the undifferentiated types of sensation as prick pain offers a situation which needs some consideration as to Head's theory of epicritic and protopathic sensibility. In the light of the extensive overlap to pain, as compared to touch, the theory of dual innervation must again be scrutinized. It is a fact that immediately after injury the overlapping sensory nerves do not begin to functionate until two, three or more weeks have

elapsed. After the sensory function has once been taken over by the overlapping nerves, resection and suture of the injured nerve produces practically no change in this area.

Any return of sensation in the area of a possible overlap cannot be accepted as a sign of regeneration. It is entirely possible that the return to prick pain in the area not included in the overlap returns only with the sense of touch, and what was heretofore interpreted as the return of protopathic sense in a nerve, was only the assumption of function of an overlapping nerve.

BOSTON SOCIETY OF PSYCHIATRY AND NEUROLOGY

Regular Meeting, Oct. 16, 1919

GEORGE A. WATERMAN, M.D., *President*

PUNCTURE OF CISTERNA MAGNA IN MENINGEAL BLOCK.

Presented by DR. JAMES B. AYER.

During the past few years, there have been brought forward, particularly by French writers, a number of methods designed to reach the cerebrospinal fluid spaces when lumbar puncture fails because of meningeal block. The principal methods employed have been puncture of the subarachnoid space at thoracic and lower cervical levels, also puncture of the basilar cisterna by introduction of the needle through the sphenoidal fissure, and puncture of the lateral ventricles. The usefulness of these procedures varies, of course, with the seat of obstruction.

In June, the writer with others (Wegeforth, Ayer and Essick, Am. J. M. Sc. **47**:789, 1919), published the technic for performing puncture of the cisterna magna, by introduction of a hollow needle through the occipito-atlantoid ligament. This procedure was found to be easy on the cadaver. By injection of fluid blackened with carbon, free communication was found to exist between the cistern and the cerebral and spinal subarachnoid spaces. This puncture was therefore advocated as a method of tapping the upper fluid reservoirs in cases of blocking at any point in the spinal canal.

The writer has recently had occasion to put cistern puncture to a practical test in such a case. The patient was first seen after meningitis had been present for about five weeks. Lumbar puncture yielded 6 c.c. only of clear yellow fluid which clotted entirely in a few minutes and which contained a large amount of proteid, only a few cells and no bacteria, either in film or culture. Puncture of the cisterna yielded 20 c.c. (more could easily have been obtained) of white purulent fluid under high pressure (400 mm. by manometer) containing a large number of polymorphonuclear leukocytes and gram cocci. On culture the organism was identified as *Staphylococcus aureus*. The lumbar fluid, presenting the Froin syndrome, together with the absence of organisms, may be considered as conclusive evidence of blockage of the spinal subarachnoid space at some point; the cistern fluid unquestionably drained infected cerebral meninges.

Two subsequent cistern punctures have been performed in this case, the fluid on the second occasion being much less cloudy and on the third almost clear, with few cells and no bacteria in film or culture. In spite of this fact, however, the patient now presents symptoms probably referable to internal hydrocephalus, and it is likely that ventricular infection still persists, for which puncture of the lateral ventricles will be required.

The case is presented because it clearly shows the availability of cistern puncture in spinal subarachnoid block. The puncture was performed with ease on all three occasions, and with no ill effects to the patient at the time or subsequently. On the first occasion ether was given; the other two punctures were made under procain anesthesia.

MEDITATIONS ON MORALE. Presented by Dr. Donald Gregg.

Dr. Gregg defined morale as a state of mind or consciousness conducive to the control of conduct by intellectual ideals rather than by instinctive impulses, and pointed out the various instincts which have been studied in their effect on human conduct as expressed by Bergson, Jung, Adler, Freud, Trotter and Sidis. He brought out the fact that two fundamental instincts, the gregarious and the self-preservative, have been of especial importance during the war in relation to morale. Inheritance, acquired disabilities and environmental strain he regarded as the factors conducive to the augmentation of the instinct and reactions, and pointed out how each of these played a definite part in individual development. He spoke further of the morale division of the army whose chief effort had been to augment intellectual ideas and to remove or mitigate as far as possible conditions that beget ill health, anxiety and fear.

If we could plot out our intellectual ideas and our instincts, Dr. Gregg queries whether we would not find that our morale would be represented by the separation between the two curves, and whether with a fall in ideals or with a rise of instinctive reactions the margin of morale would not decrease. He concludes that without morale an army is ready for defeat and rout, and an individual is ready to develop psychoneurosis.

MEDICAL AND SOCIAL ANALYSIS OF DATA OF OUTPATIENT DEPARTMENT, PSYCHOPATHIC DEPARTMENT, BOSTON STATE HOSPITAL. Presented by Dr. A. Myerson.

Psychiatry was born in the asylum and up to the present time has carried the point of view therein engendered. The central conception of insanity which is essentially a legal matter has tremendously hampered the psychiatrist, nor does the substitution of the word psychosis help matters. The outpatient psychiatrist takes this point of view: he deals with mental disease and includes in this category on an even basis the so-called psychoses and the so-called psychoneuroses. Concerning the former, it may be stated that not every dementia praecox patient is insane, nor is every patient afflicted with general paresis insane. Concerning psychoneuroses, every case of psychasthenia and every case of hysteria is a purely mental disease, and the neurosis part of the title is a shadowy euphemism. Some psychoneurotics need to be committed to an insane hospital though the majority do not. Commitment is to be regarded as a social and individual therapeutic measure, and has no bearing on diagnosis or classification. All cases coming before the psychiatrist and diagnosed as mental disease bring up the question of therapeutics, and among these commitment is only one of the therapeutic possibilities in psychiatry. It may be necessary to utilize it in a case of dementia praecox, and it may be necessary to utilize it in a case of anxiety neurosis.

An effort was made to analyze the statistics of the outpatient psychopathic department of the Boston State Hospital with the following questions in

mind: 1. Who sent the patient? 2. What problem does he present? 3. What diagnosis is made? 4. What is done with him? The answers to these questions throw light on the social and psychiatric problems of the community.

The largest individual group of cases came from social agencies, and their interest was greatest in the female adolescent; next, in the female adult; male children were third in importance; female children fourth, and the male adolescents were given scant attention in so far as their mental state is concerned. The principal problems brought for solution by the social agencies were delinquency, especially of the sex type and illegitimate maternity. Of the large variety of diagnoses made in the social cases, mental deficiency took first rank in point of numbers. Psychoneuroses were relatively uncommon and various psychoses occurred fairly frequently. A large percentage of the cases had no disease that could be diagnosed; in other words, were merely suspects. Interesting relationships were found in the case of other sources from which the patients came, such as the courts, schools, positions, other hospitals, own initiative and the psychopathic department wards. Each source of patients was individual in the type of cases sent and in the problems presented. The psychopathic outpatient department is a consulting house for diversified groups of people, each one of whom is individual in the problem presented.

HISTORICAL CAUSES OF THE PSYCHONEUROSES OF THE JEW.

Presented by DR. A. MYERSON.

Among various matters considered important in the problems presented in neuropsychiatric clinics is the preponderance of Jewish psychoneurotics. The historical background of the Jew was searched for causes, as has often been done before. It is contended that before biologic heredity can be evoked as a cause of racial predisposition and liability to disease, what is termed social heredity must first be analyzed and examined. Social heredity is a group of life conditions and opinions handed down from generation to generation and of extraordinary potency in molding the character of a race.

In the case of the Jew this may be briefly summarized as follows: Owing to the restrictions and limitations made by his Christian "hosts" he was excluded from the soil and debarred from handicraftsmanship. Thus his manual motor instincts were suppressed. The large basal movements by which psysique is built up and nervous tension discharged were made inaccessible and he became almost exclusively an urban dweller, excessively cerebral in his pursuits, despising exercise and sports, and thereby lowered his physique and endurance. Due to the precariousness of his social and economic position, the threshold for fear was lowered; due to the close family life and the close racial life, an unhealthy emotionality was engendered, though this family life and clandishness in part accounted for the survival of the Jew. Introspection came easily to a race of cerebral workers, excluded from sports and very apprehensive. These factors may explain the psychoneuroses of the Jew.

That social heredity is easily cast off under freer conditions may be seen at the present time in Jewry. The close and pure family life is becoming a thing of the past. Antipathy to sports and the poor physique therein resulting, tends to disappear in the second generation. The Jewish prize fighter is as common and as successful as the Jewish scholar. The overvaluation of money can hardly be exemplified in these sons of the pious Jew who now are

the greatest sports in America, and obnoxiously so. Social heredity differs from biologic heredity in the quick changes that may occur once the link with the past is broken, and this seems to be occurring at the present time in the case of the American Jew.

DISCUSSION

DR. DONALD GREGG asked whether Dr. Myerson had any theory as to the basis of the instinctive dislike of one race for another. That is to say, inasmuch as various races have varied immunities to disease, there may very possibly be an instinctive objection to another race on a biologic basis, if that race is subject to certain diseases that the first race is not. For instance, the negro has a different immunity to tuberculosis and syphilis from the Anglo-Saxon. Is it possible that the instinctive feeling that the Anglo-Saxon has toward the negro is based on such biologic facts?

DR. E. W. TAYLOR asked Dr. Myerson whether there were any statistics or facts available relative to the part the Jews had taken in the war. Of course, they entered the war in large numbers and his impression was that they stood up under it equally with any other group, or class or race.

He spoke in strong commendation of Dr. Myerson's opening remarks on the use of terms, particularly on the use of the term insanity and especially the confusion of the legal with the medical aspect of the term and agreed with him that the term psychoneurosis is a misnomer, that the neurotic element implying structural change is tremendously in abeyance and is nonexistent in a great many instances. Hysteria, for example, is a true psychosis. It is, however, useless to enter into a discussion as to the terminology as this is one of the greatest difficulties in clarifying medicine in general and neuropsychiatry in particular. The point he would make was that it was desirable to study each case carefully and to use terms as far as possible in a general rather than in a too specific way.

It is regrettable that there is a feeling on the part of the community that the psychopathic hospital is still a hospital for the insane. However, the outpatient department has done, and is doing, a great deal to overcome this feeling and all the agencies, physicians included, should be more generous in sending patients to the psychopathic hospital in order, among other things, as far as possible to overcome this still prevalent feeling. The greatest possible advance in the last few years has been to recognize the predominant mental character of a great number of the conditions dealt with.

DR. WALTER CHANNING expressed himself as delighted with Dr. Myerson's paper in that he had summarized the great advance that has been made in the last ten years in the consideration and treatment of the whole subject of mental diseases. We probably can never have an exact classification of such diseases because the mental states are really without number, and one may to a certain extent run into another so that we can only in a general way describe them. Every time we hear any one speak of the use of the word insanity as did Dr. Myerson and Dr. Taylor, we are making progress. The word has become merely a social term, meaning that a person subject to mental disease is regarded as a proper case for an institution, and that it is proper to commit him. In the last analysis the most important outcome of the psychopathic hospital will be the outpatient department. A great

many years ago, a little department of the Boston Dispensary was started, but patients almost had to be paid to go there. Even at that time the Jews constituted a large part of the patients. They seemed to be more intelligent and more willing to come and place themselves under treatment without undue sensitiveness about being called possible cases of insanity.

Dr. Alfred T. Noyes called attention to the fact that Dr. Myerson had, by means of tables (which were shown), presented very concrete facts as to where the patients came from, what the diagnosis was, what the problem presented and what was to be done for them. Heretofore at the psychopathic department there has not been a concrete arrangement in just this manner. Due largely to Dr. May's instigation, Dr. Myerson has evolved this scheme, and this is the first year in which it has been possible to apply it. If this is applied for say fifteen years, some very practical statistics will result.

Dr. Myerson in closing in answer to Dr. Gregg's question as to whether or not an antipathy of an instinctive kind exists in races, thought this possible. It is certainly true that perception of unlikeness in any gross way arouses an instinctive dislike. Unlikeness arouses dislike. The feeling that we are just right makes us antagonistic to one unlike ourselves. This is noticeable in children. In the movies, for example, when pictures of foreign peoples are shown a titter is apt to run through the audience. Even though in themselves pleasing, nevertheless differences in custom and costume arouse an instinctive dislike. It requires education and study to recognize that what is unlike ourselves possibly is as good as we. This is the process of education. As to whether there is an antagonism on the basis of disease, he expresed no opinion. Other far more palpable unlikenesses exist. The negro is disliked because of his black skin. The Jew is different and therefore the subject of dislike.

Dr. Taylor inquired about the army. The statistics of the army are very good. There are less than 3,000,000 jews in the United States, most of them on the Atlantic seaboard. The total population of the country is over 100,000,000; a little under 3 per cent., therefore, are Jews. The number of Jews in the army was over 4 per cent., an increase of one third over the allotment. There were a great many Jews who objected to serving. The native born Jew was as willing as any one to enter the army. The foreign born Jew, on the other hand, largely Russian, showed for a long time antipathy to to the war largely because he was at first pro-German for the reason that Russia was on the other side. Consequently, he rejoiced when the Germans beat the Russians. When the United States entered the war the situation changed. The fatalities and casualties among the Jews were as large in proportion to their numbers as those of other races.

Concerning the general subject of insanity, he thought the critical point was that we are always getting into difficulties when we make an abstraction from a reality. Insanity is an abstraction. We are dealing merely with mental disease. Given a mental disease—hysteria, psychoneurosis, dementia praecox, anything you like—it may or may not be a committable case. The larger number of general paretics become committable, but not all paretics are insane or committable. On the other hand, the majority of hysterics are not committable but there is nothing in hysteria to prevent such a step from becoming necessary; similarly with psychasthenia. Commitment is merely a legal and therapeutic step, not at all diagnostic; at present it is locked up with "insanity" and psychosis and these terms should be dropped.

PHILADELPHIA NEUROLOGICAL SOCIETY

Regular Meeting, Oct. 24, 1919

J. HENDRIE LLOYD, M.D., *President*

CASE OF A EUNUCH CONVICTED OF RAPE. Presented by DR. N. S. YAWGER.

This presentation by Dr. Yawger showed the retention of sex desire to a felonious extent many years after complete extirpation of the gonads.

A colored man who was sexually active at 18 years sustained a severe injury by being struck by a locomotive. He was knocked astride a switch-post thereby fracturing his skull and right leg and badly lacerating both testicles; the latter were subsequently completely removed. The man lay unconscious for five days and then recovered. As a result of this injury, according to his statement, he differed from his former self only in the loss of about 30 pounds in weight, but that in particular he was absolutely without sexual power and sexual desire.

In December, 1917, a white woman charged that he had committed rape on her. The man was indicted, tried and found guilty. He was then 42 years of age. The fact that he had been castrated 24 years before was not brought out during the trial, but this condition was the prominent feature of the request for a new trial. The case was not reopened, and a sentence of from ten to fifteen years in the Eastern State Penitentiary was imposed. Shortly following the convict's entrance into the prison, an interview was sought with Dr. Yawger when the above facts were disclosed, and he repeated emphatically that following castration there had been neither sexual power nor sexual desire. The prisoner then asked, since it was impossible for him to have committed rape, whether he should not be pardoned?

Examination of the castrate showed him to be rather thin, flabby and in appearance several years older than his stated age; the penis was easily of average size. Dr. Yawger believed in regard to his sexual feeling and capacity that the prisoner had prevaricated, and told him so.

Literature bearing on this subject is interesting. Breeders of stock tell us that the physical appearance and nature of a male undergoes a vastly greater change when castrated young than when this operation is performed on the adult; and that the sex desire is especially likely to remain if the animal has previously been standing at stud. Formerly, at the earnest solicitation of parents, surgeons occasionally castrated boys afflicted with feeblemindedness and dementia praecox with the thought that masturbation would be overcome and in the hope that mental improvement would then ensue; some of these boys, on the healing of their wounds, resumed their pernicious habit and none were advanced to normal mentality. That considerable sexual power may persist in a eunuch, appears to have been recognized, since we are informed that the mutilation of males attendant on harems was frequently carried beyond castration—sometimes the penis was amputated.

A CASE SHOWING JACKSONIAN ATTACKS (STATUS EPILEPTICUS) WITH NECROPSY. Presented by DR. SAMUEL LEOPOLD.

Dr. Leopold said that epilepsy of syphilitic origin is not an infrequent picture, but this patient is of interest because she showed a status epilepticus and because there was an opportunity to study the pathologic process.

A colored woman, aged 38, twelve years ago had fits in which she would run around the house and, according to the sister, would tear off her clothes. These attacks occurred at irregular intervals until her admission to the hospital.

For several days before admission to the hospital she had had several convulsions a day. She was admitted to the hospital during one of these attacks which ceased half an hour later. The next morning, at 9 o'clock, the convulsions began again and continued constantly until she died at 11 o'clock that night. The attacks were limited to the left side of the face, left arm and leg, and were clonic in character. There was conjugate deviation of the eyes to the left, and the head was turned to the left.

The convulsions started in the face, then involved the neck, arm and leg on the left side; occasionally there was a slight movement in the right ankle and foot. The patient was unconscious; the movements were slow and coarse. The pupils were contracted and did not react to light. A study of the reflexes was not satisfactory, all seemed to be lost. Heart and lungs were negative. Wassermann reaction of the spinal fluid was positive.

At necropsy, the brain showed a marked gummatous meningoencephalitis which involved the right frontal lobe and extended to the precentral convolution.

A CASE OF ESSENTIAL EPILEPSY EXHIBITING SPECIAL FEATURES AND GREATLY IMPROVING UNDER OPERATIVE PROCEDURE. Presented by Dr. Alfred Gordon.

A male, aged 32, developed at the age of 12 epileptic seizures of a generalized character. Until six months ago the attacks occurred very frequently, from two to four daily, with short intervals of intermission, the longest of which was one week. Six months ago, when the man first came under Dr. Gordon's observation, he had had an unusually severe attack. After recovery a distinct aphasia was observed. It was of a pure motor type (aphemia) in which the inner speech was preserved. There was no paralysis of the extremities, and no indication of the involvement of the motor area or its tracts. The reflexes, deep and superficial, were unaltered.

The eye examination was negative, with the exception of irregularity of the right pupil. This, according to the ophthalmologist, was of no significance.

The examination of the urine, the Wassermann test on the blood and spinal fluid and the roentgen-ray investigation of the skull were all negative. There was no history of venereal infection.

In view of the clearly defined motor aphasia, the presumption was that a hemorrhage probably took place over Broca's convolution. On the thirteenth day following the supposed hemorrhage, the aphasia remained unaltered, contrary to what is usually observed in cases of aphasia occurring in the course of epilepsy. In the majority of instances the aphasia is temporary and transient, comparable to the paresis of one or two extremities which sometimes occur in epilepsy. These phenomena are all due to a temporary inhibition of the corresponding centers and of their tracts.

In view of the persistence of the disorder, a lesion in the form of a hemorrhage was considered. Operation was advised and promptly accepted by the patient. An osteoplastic flap was removed from the left side of the skull over the frontoparietal region. A hemorrhagic focus was found over the lowest portion of the third frontal convolution.

Rapidly the patient's speech commenced to improve, and in three or four weeks returned to almost normal. For four subsequent months there was no convulsive seizure. Due to some indiscretions in sexual matters and to an enormous meal on the same evening, he became ill and had his first convulsive seizure since the operation. His speech again became affected. During the last two months there had been no epileptic attacks. The patient showed some disturbance in speech, but no other abnormal condition. The case was interesting from several standpoints: (1) The occurrence of a hemorrhage during an epileptic attack over Broca's region producing a persistent motor aphasia. The condition was very exceptional. (2) Arrest of persistent convulsive seizures of a violent character following an operative procedure on the skull. (3) A practical question arises as to the advisability of performing a cranial decompression in essential epilepsies even without focal and well localized symptoms (that the intracranial pressure in all epilepsies is elevated, is a well-known fact).

DISCUSSION

Dr. William Spiller said that a child had been brought to the dispensary of the University Hospital recently whose condition was similar to that in the case presented. The child was about 15 years of age; it had had general convulsions most of its life, and four years ago had become hemiplegic. Dr. Spiller thought that possibly the hemiplegia had developed as a result of the convulsions, but the paralysis had been of too long duration to permit operation. He recalled a case of hemorrhage into the conjunctiva, occurring during epileptic convulsions. Where there was congestion, such as occurs in repeated epileptic attacks, there might be a rupture of a blood vessel in the brain.

The fact that Dr. Gordon's patient had had no attack for four months after the operation did not necessarily mean that the man would have no more convulsions. There may be benefit from an operation on the brain in epilepsy, for a time, even when nothing has been removed.

Dr. Spiller thought Dr. Gordon's patient did not have aphasia. His speech was very indistinct, but there was no true aphasia.

Dr. J. Hendrie Lloyd said that he recalled Dr. Gordon's case. The patient was under his care at the Philadelphia Hospital. He was surprised to see him put down as a case of essential epilepsy, with aphasia. Dr. Lloyd thought the man had some speech defect more like a dysarthria. He thought this point important because it antedated Dr. Gordon's first observation of the case. His recollection of it was that it was not an aphasia, it was more like a dysarthria. It was an interesting point and that was the reason he dwelt on it.

Dr. Alfred Gordon said that he was surprised to hear that the patient had been in the Philadelphia Hospital. At the time he saw him first, in April of this year, the history was given to him just as he had reported it, and following his last attack, which he had had a few days before Dr. Gordon saw him, he developed this aphasia. Formerly he spoke perfectly well. That was the testimony of his father, mother and sisters. Then the operation revealed the hemorrhage over the lowest portion of Broca's convolution and the ascending frontals. The operation was beneficial. The surgeon removed the blood, washed it out and the cortex appeared quite clear. The operation was performed ten or eleven days after the attack; the patient began to talk a few days later. The inhibition of the parts corresponding to the speech center persisted to a degree, but he made rapid progress, so much so that the aphasia was considered quite cured. A new attack occurred and then the speech again

became disturbed. Perhaps he had a second hemorrhage. He refused a second operation, which might have improved his speech.

A CASE FOR DIAGNOSIS. Presented by Dr. S. F. Gilpin.

A white male, aged 62 years, a carpenter by occupation, fell 6 feet into the hold of a ship in November, 1918, which caused the present condition. The man was rendered unconscious, and was taken to a hospital. The roentgen-ray examination at the hospital showed a fracture of the orbital plate of the frontal bone. An operation was not performed. The patient was unconscious for at least four hours. While in the hospital he complained of pain in his arms, and he was unable to raise them or to sit alone. During this time he had incontinence of urine. He remained in the hospital sixteen days, and was taken home on a stretcher, being unable to use his arms and feet He was out of bed in five weeks, and dragged his right foot in walking. The control over the urinary sphincter returned about this time, but he lacked perfect control of his bowels. He was fed by his daughter for three months after his discharge from the hospital. A tremor developed during the past three months.

Examination, Oct. 11, 1919, showed that the pupils reacted promptly, station showed some slight increase in sway. The man arose from a chair and sat down stiffly. His gait was a shuffling, hesitating one; he scraped his right foot along the floor and had a tendency to turn toward the right. A coarse general tremor was produced when he attempted to walk; this tremor was most marked in the hands. He carried his upper extremities in the position of a case of paralysis agitans. His grip was weak in both hands, but equally so. The sphincters were controlled. All motion was slow and stiff. He assisted himself with his hands when crossing his legs. The knee jerks were rather exaggerated; there was no Babinski and no foot clonus. There was no sensory disturbance. The mental condition was normal, except that he was emotional; at times he broke down and cried. He was given compensation at the rate of $10 per week for four hundred weeks.

The case was an important one from the practical standpoint of diagnosis in cases requiring compensation. Was this a case of paralysis agitans? The patient's muscular weakness and rigidity of attitude in turning suggest this disease. However, the tremor came out on attempting motion, and was especially marked when the patient knew he was being observed. When climbing stairs he showed no tremor. If he had paralysis agitans, was the disease developing before the trauma or was the trauma the direct cause? His gait and general bearing suggested hysteria. If hysteria was the explanation of his case he should be cured and the compensation adjusted. It would seem plausible to ascribe his symptoms to the severe concussion his brain must have suffered to produce the symptoms immediately following the accident.

DISCUSSION

Dr. F. X. Dercum said that the attitude of the patient and his manner of turning around strongly suggested paralysis agitans. There was no tremor, however, except just as the hands came to rest, but tremor is occasionally absent in paralysis agitans. However, the fact that the man was a "compensation case" and was at present receiving compensation, which was to extend over a number of years, must be taken into consideration. It was found that very strange symptoms were every now and then presented by litigation and compensation cases, symptoms which did not subside until the

case was settled in litigation cases or when the payments ceased in compensation cases. Of course this case was perhaps a genuine case of paralysis agitans and not one of hysteria, and it was possible also that the man may have been developing paralysis agitans when he received his fall. The weight of the evidence, however, Dr. Dercum thought, was in favor of hysteria.

Dr. J. Hendrie Lloyd said that as Dr. Gilpin raised the question of hysteria, he would say that the gait looked very much like the gait of astasia-abasia. He had, however, never seen a case of astasia-abasia in a middle-aged man; it usually occurred in young women.

Dr. William G. Spiller said astasia-abasia did occur in men of middle age, and he had recently had such a case. There have been many cases in which trauma has been supposed to be the predisposing cause of paralysis agitans.

Dr. Charles K. Mills said the patient looked to him like a man who had had paralysis agitans, and then had a traumatism which resulted in hysterical astasia-abasia.

Dr. Alfred Gordon said that this man had a fracture of the skull. It was demonstrated by the roentgenogram. If it was a question of speculation, why not consider other possibilities, such as involvement of the lenticular nucleus? In diseases of the lenticular nucleus some features of the present case have been observed. The patient may have had a hemorrhage into the lenticular nucleus by contre-coup. As far as the astasia-abasia or paralysis agitans were concerned, the patient's condition was not typical of paralysis agitans nor of astasia-abasia. Dr. Gordon said he believed in the possibility of a real organic lesion in this case.

Dr. S. F. Gilpin said that he had seen astasia-abasia in middle-aged men. The marked general tremor when the man tried to walk, and again, when he was told to control it, his ability to control it, and further, Dr. Gilpin said he had watched him climb stairs to the second floor without a sign of tremor. Evidently some of these symptoms were controlled more or less. Dr. Gilpin stated that the roentgen ray did not show fracture at the present time. He did not see why the condition in a man whose nervous system had been shocked as had this man's, should not have an organic basis.

A CASE OF POSTEROLATERAL SCLEROSIS ASSOCIATED WITH ANEMIA. Presented by Dr. Joseph McIver.

A white male, aged 53, was admitted to the service of Dr. James Hendrie Lloyd, at the Philadelphia General Hospital, September 2 of this year. Dr. McIver's principal reason for bringing the case before the society was the fact that syphilis had been suggested as a possible etiologic factor.

About April, 1918, the patient complained of an aching pain in the left shoulder, which had continued at varying intervals to the present time. Some time later he noticed paresthesias in the tips of the fingers of both hands. This symptom had continued and had steadily grown worse. In the early part of this year he noticed that his legs were becoming weak, and he had to give up his work as a laborer. Soon he began to suffer from paresthesias in the lower extremities. The condition had slowly progressed and since September 2 he had been confined to bed.

The family history was negative. The past medical history was likewise negative except for gonorrhea twenty-five years ago. He denies very emphatically any syphilitic infection. He complained of weakness and paresthesias in both lower extremities. He stated that his heels felt as though the bones were

going through the flesh. There was also a stinging sensation in the hands and feet. There was a history of incontinence of feces on one occasion.

Physical Examination: The pupils showed a slight inequality, but reacted well for light and in accommodation. The eye grounds were normal. The teeth were in very bad condition. The biceps and triceps reflexes were present and normal. There was a slight degree of ataxia in the finger to nose test, but there were no sensory disturbances in either of the upper extremities.

The lower limbs were spastic, and there was a marked Romberg sign. There was marked ataxia in gait and in the heel to knee test. The achillis tendon and patellar tendon reflexes were markedly exaggerated; the Babinski reflex was present on both sides. Tactile, pain and temperature senses were normal throughout. The sense of position and vibratory sense were lost in both lower extremities. The urine was negative. An analysis of the gastric contents showed free hydrochloric acid 17, total acidity 50, and lactic acid present. The examination of feces for ova and parasites was negative. Blood Wassermann was negative. Spinal fluid Wassermann on one occasion was ++ with a cholesterinized antigen; and on two occasions was anticomplementary. In discussing this report with the serologist, Dr. McIver was convinced that very little positive significance could be attached to it. Cell counts of the spinal fluid were 30, 10 and 20 on various occasions.

Blood Count: Hemoglobin, 60 per cent.; erythrocytes, 2,229,000 per cubic millimeter; leukocytes, 7,000 per cubic millimeter; color index, 1.3. Differentials: polymorphonuclears, 68 per cent.; small lymphocytes, 18 per cent.; large lymphocytes, 7 per cent.; eosinophils, 7 per cent.; basophils, none. Abnormal erythrocytes: few metachromatic, few microcytes, few macrocytes. Nucleated erythrocytes not found. Abnormal leukocytes not found except an excess of eosinophils. Blood diagnosis: secondary anemia, pernicious in type.

Dr. McIver said that in combined sclerosis of the spinal cord associated with anemia, the degeneration of the posterior column was peculiar in that it began in the more medially situated fibers, that was to say, in the long fibers of the columns of Goll. Consequently in the early stages, deep sensation alone was disturbed, more particularly, bone sensation and the sense of muscular position. In April, 1916, Dr. William G. Spiller reported to the Philadelphia Neurological Society the microscopic findings of the spinal cord in a case of tabes associated with severe anemia. He was able to demonstrate, not only the usual lesions of tabes dorsalis, but also the characteristic alterations of the spinal cord seen in anemia.

So far as Dr. McIver had been able to discover, these particular sensory phenomena did not occur as isolated signs of syphilitic disease, but were associated sooner or later with a type of anemia that was fatal.

Preservation of pain and tactile sensations with increased tendon reflexes was the rule in the early stages of combined sclerosis associated with anemia. These sensations might be affected late in the course of the disease with diminution or loss of the tendon reflexes. In cases of syphilitic posterolateral sclerosis, pain sense was disturbed first because the disease began as a meningitis and did not begin in the cord proper.

DISCUSSION

Dr. J. Hendrie Lloyd said that this was a case in which Dr. McIver and he had been very much interested. There had been some differences of opinion expressed in regard to the causation. The patient's appearance hardly supported the diagnosis of pernicious anemia. It was a secondary anemia not

pernicious in type. But the patient had a double plus Wassermann; he had the symptoms of a posterolateral sclerosis, as had been described, and he had no pupillary involvement indicative of syphilis. The idea of pernicious anemia causing cord changes was of course not a new one. Dr. Lloyd had reported more than twenty years ago one of the best marked cases occurring in the Philadelphia Hospital. Recently, the subject has been much exploited. An author in the West said he has seen nearly three hundred cases of pernicious anemia in three years, which is certainly an extraordinary record. Dr. Lloyd said he himself had not seen three hundred cases in thirty years, or a number anywhere near that. In Minnesota they had the bothriocephalus latus, which caused an anemia very similar to pernicious anemia. The author in the West does not mention this; nor does he give a blood count or a Wassermann test, so Dr. Lloyd thought that altogether he had written a rather unsatisfactory and unscientific paper. The bothriocephalus tapeworm is a tapeworm that we do not have in this country unless it is imported. It prevailed more particularly in Scandinavian countries, and had been brought to Minnesota, where they had Scandinavian immigrants. Dr. Lloyd thought that some of those cases of so-called pernicious anemia in the West that were reported from Minnesota and Wisconsin were probably cases of Bothriocephalus anemia. Bothriocephalus anemia is like any other anemia of high grade in that it might possibly cause changes in the cord. Dr. McIver's case, however, had shown no symptoms of any tapeworm infection. Dr. Lloyd had once seen a case of Bothriocephalus infection, with a high grade of anemia, in a young Danish woman in Philadelphia. The anemia was of the pernicious type, and yet the patient had no marked symptoms of involvement of the spinal cord. In the present case he has not felt satisfied with the diagnosis of pernicious anemia, because the blood count and the appearance of the patient hardly supported it; and, on the other hand, the possibility of syphilis cannot be ignored. Moreover, a secondary anemia may be caused by syphilis.

Dr. William G. Spiller said that Dr. McIver brought up the subject as to whether syphilitic posterolateral sclerosis could produce the signs of a posterolateral sclerosis of anemic origin. He presumed it could if the involvement of the posterior columns was a meningitis with cellular infiltration into the posterior columns without involvement of the posterior roots, but this was not likely to occur.

The ++ Wassermann reaction in Dr. McIver's patient was no proof that the changes in the spinal cord were syphilitic. A syphilitic man might have posterolateral sclerosis from anemia, and the findings in this case were typical of the anemic posterolateral sclerosis. The man had about 2,000,000 red blood cells. Dr. Spiller said he had had a case recently in which the hemoglobin was 71 per cent. and the red blood cells were 3,700,000; within two weeks the hemoglobin had fallen to 21 per cent., and the red blood cells to less than 2,000,000, and the man died.

Dr. F. X. Dercum regarded the case as in all probability one of anemia. It must be remembered, however, that in the lesions of the cord which gave rise to the syndrome of ataxic paraplegia there was an infiltration of the membranes of the posterior and lateral aspects of the cord and at the same time a marked interference with the lumen of the vessels, so that a degree of anemia was mechanically established in the portions of the cord affected. It was not improbable that this local interference with nutrition was the dominant factor in the production of the symptoms.

Dr. Charles K. Mills said that he had a private patient under observation in which this very question was more or less at issue. When Dr. Wilson and Dr. Mills first saw the patient three years ago, they made the diagnosis of an advancing posterolateral sclerosis of anemia. At that time the Wassermann test was negative. Dr. Mills did not see much of the patient until perhaps, two months ago, when he returned. He had since been under Dr. Mills' care, and had been seen somewhat frequently. His case had advanced very markedly when he returned; he was scarcely able to stand. Three years ago he had had increased reflexes, clonus and a Babinski reflex. Strange to say, now he has lost knee jerks and lost ankle jerks, and in that respect at least, the case had changed. Dr. Mills had had two Wassermann blood examinations made since his return; one of them had proved negative, and the other positive. It appeared to him that his own case was distinctly one of posterolateral sclerosis due to anemia, but the patient also may have had syphilis.

Dr. George Wilson said that he had carefully examined and questioned the man whom Dr. McIver had presented. He did not believe that the patient had real lancinating pains, which were so common in syphilis of the spinal cord. The man had told Dr. Wilson that the pains which he complained of were due to the involuntary jerkings of the lower extremities. Subjectively the patient had various paresthetic sensations in the hands and feet, a condition that was always found in the posterolateral sclerosis seen in anemia. There was no involvement of the bladder. The sensory loss was typically that described by Williamson and later by Dejerine in the combined sclerosis seen in anemia and other conditions such as pellagra. When syphilis produced a posterolateral sclerosis, pain sense was lost, diminished or retarded, exactly as in tabes, and the sensory loss which this man presented was not found.

Dr. Wilson said that the severity of the anemia in a given case had nothing to do with the possibility of that case developing cord changes. Three years ago Dr. Wilson examined in Dr. Mill's office a man with combined sclerosis of the anemic type who had 82 per cent. hemoglobin and 4,000,000 red cells. Careful examination of a blood smear revealed two nucleated red cells. A few months ago Dr. Wilson observed for some time a most extraordinary case of pernicious anemia in the service of Dr. A. A. Stevens in the Philadelphia General Hospital. That case had 10 per cent. hemoglobin and 225,000 red cells and yet presented no nervous findings, and today with a vastly improved blood picture showed no changes in reflexes, sensation or muscular power. The anemia did not produce the cord changes but rather the same toxemia which produced the anemia caused the combined sclerosis.

Dr. Wilson said that he believed that the ++ Wassermann with the cholesterinized antigen was not worth considering, at least he had been so informed by serologists. Even though the patient had had a positive Wassermann reaction by the classical test, that could not change a diagnosis which to Dr. Wilson's mind was certain. The Wassermann test had led many astray; a great many cases with positive clinical evidences of syphilis were allowed to go untreated because the Wassermann reactions were negative and other cases with positive Wassermann reactions were not investigated further because they had a disease which might be present without being the cause of the syndrome found. As a case in point Dr. Wilson recalled a specimen of a spinal cord showing a tumor which Dr. Lloyd presented to the society. The Wassermann test was positive, but the tumor was beyond the syphilitic pale.

Dr. C. S. Potts said he thought, from what Dr. Lloyd had said, that his reason for not attributing the symptoms to anemia was that the blood did not

show the picture of pernicious anemia. As Dr. Potts understood it, the condition could be caused by any form of anemia, and if he remembered correctly, a patient had been shown to this society several years ago in which the symptoms followed anemia caused by a severe hemorrhage. Chronic diarrhea and other exhausting conditions and toxemia have also been stated to be causes. The history and symptoms seemed in this case to be very characteristic of the so-called combined sclerosis which may follow pernicious and other forms of anemia.

Dr. J. H. W. Rhein said that he had a woman under his care at present who presented typical symptoms of posterolateral sclerosis, in whom the symptoms were practically identical with those seen in the case presented by Dr. McIver. The symptoms of the spinal involvement appeared when the blood picture was that of a secondary anemia. There were 3,700,000 red blood cells and 65 per cent. hemoglobin and an absence of anisocytosis and poikilocytosis. The color index was 1. In three weeks' time, however, the hemoglobin went to 60 per cent. and there were 1,910,000 red blood corpuscles present with some poikilocytosis and anisocytosis. Then under medication the blood picture became less like pernicious anemia, there being 3,720,000 red blood cells present and the hemoglobin advancing to 75 per cent.

He recalled a somewhat similar case which he had had under his care in the Polyclinic Hospital—similar to this one as far as the blood picture was concerned—and which presented the typical symptoms of posterolateral sclerosis without the typical findings in the blood of pernicious anemia. He commented on the fact that he had recently seen and heard of a few cases in which posterolateral sclerosis developed in anemias which were suggestive of, but not typical of, pernicious anemia.

It was possible, therefore, for the spinal cord changes to be quite far advanced before the blood itself showed the typical features of pernicious anemia, and while students of blood conditions will not admit that cases of this sort were really pernicious anemia, it was Dr. Rhein's belief that they belonged to the same group of blood diseases.

Dr. Alfred Gordon said that if we considered the general histories of syphilis of the cord, in the majority of cases in addition to the tract involvement there was involvement of the sphincters which was absent in the present case. He did not see why we hesitated to accept the possibility of anemia when the blood picture was typical of anemia; hemoglobin 60 per cent., and the red blood cells greatly decreased in number. The picture of anemia was clear. The presence of syphilis, as judged by the spinal fluid examination, was exceedingly doubtful. The posterolateral sclerosis of anemia was not such a rare thing. It had been reported, and personally Dr. Gordon did not feel any hesitation in accepting the diagnosis of posterolateral sclerosis due to anemia.

Dr. Joseph McIver said that he had always regarded the case as one of rather severe anemia. Whether or not it was a pernicious or secondary anemia, could only be settled by the blood picture. This had been done entirely to his own satisfaction by Dr. Rivas who had made the blood examination. He thought undoubtedly the case was one of posterolateral sclerosis such as is associated with anemia.

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